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Original Research Article

HbA1c as a prognostic indicator in prediabetics with acute coronary syndrome

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ABSTRACT

Background: The role of HbA1C in predicting the outcomes of acute coronary syndrome remains controversial. Lesser is known about it in non-diabetic patients. Therefore authors conducted a study to seek association between the HbA1C levels and the clinical outcome in non-diabetic patients who presented with acute coronary syndrome. Objective of the study was to estimate HbA1C levels in population of prediabetics and non-diabetics and to document and correlate major adverse cardiac events in prediabetic and non-diabetics.
Method: This case control study included consecutive patients (n=68) without known diabetes mellitus admitted with acute coronary syndrome (STEMI, NSTEMI, UA) at our hospital. HbA1c was measured on admission. The patients were divided into 2 groups according to their HbA1c level (group 1 HbA1c<5.7%, group 2 HbA1c>5.7%). The main outcome was MACE (major adverse cardiac events including cardiogenic shock, arrhythmia, heart failure).
Results: There was no significant difference between baseline characteristics of both groups but complications were seen in higher number cases with HbA1c >5.7%. No significant difference in mortality was found. On analysis HbA1c >5.7% was found to be an independent predictor of MACE.
Conclusion: HbA1C is a predictor of major adverse cardiac events. Measurement of HbA1C levels may improve risk assessment in such patients presenting with ACS.

Keywords: Acute coronary syndrome, HbA1c, MACE, Nondiabetic, Prediabetes

INTRODUCTION

Cardiovascular disease has been considered as the important cause of death in industrialized nations. Acute coronary syndrome (ACS) encompasses a continuum ranging from unstable angina, STEMI and NSTEMI. The important risk factors are hypertension, dyslipidemia, type 2 Diabetes Mellitus (DM), insulin resistance, obesity and cigarette smoking.

Unlike other cardiovascular risk factors, obesity and type 2 diabetes are showing a significantly peaking pattern. Uncontrolled diabetes has high incidence of ACS and poor prognosis. Higher blood sugar value during admission for ACS carries grave prognosis not only in diabetics, but also in non-diabetes patients.

Poor glycemic control have high incidence of ACS which inturn have poor outcome. Also it is seen that hyperglycemia without previous history of DM are not uncommon in patients presenting with ACS.[1] Inadequate glycemic control or management is shown by elevated HblAC, and its elevated value during admission, increases the mortality in first month. Increase in the blood sugars at the time of ACS without the history of DM has increased short term mortality. The point of fact that elevated blood sugar can be an indicator of already prevailing insulin resistance and defective function of

beta cell which can result in poor prognosis. Moreover, the stress induced secretion of catecholamine leads to partial inhibition of pancreatic β-cell release of insulin with increase cortisol and glucagon levels, leading to impaired glucose tolerance and elevated glucose levels.[2,3]

There is a rise in inflammatory markers in subjects with impaired glucose tolerance or overt diabetes which is heralded by an acute hyperglycemic event.

Following this school of thought, it might be speculated that the detrimental effect of stress hyperglycaemia in acute MI might also stem from its ability to increase inflammation.

History

Claude Bernard observed and explained acute hyperglycemic and intermediate hyperglycemia/prediabetes response to stress more than a century ago.[1]

Prediabetes and the heart

Prediabetes is the precursor stage before diabetes mellitus in which not all of the symptoms required to diagnose diabetes are present, but blood sugar is abnormally high. This phase is often referred to as the "grey area".[2]

Cardiovascular disease accounts for 70 - 75% of deaths in diabetic and prediabetic people, with acute myocardial infarction being responsible for 30%. They are at heightened risk of atherosclerosis associated disease, the contributions of the various cardiovascular risk factors are several abnormalities such as hyperglycemia, insulin resistance, dyslipidemia, hypertension, procoagulant changes and endothelial dysfunction - all appear to play important roles.

Mechanisms of hyperglycemia in acute myocardial infarction

Stress hyperglycemia [3,4]

Stress plays an important role in the regulation of insulin secretion. Acute insulin response is inhibited by catecholamines by stimulating alpha adrenergic receptors (Figure 1).

The mechanisms that operate during stress are the adrenal medulla along with components of sympathetic system help to actuate fatty acids, glucose and lactic acid.

The means by which the glucose increases is:

- In the liver there is increased glycogenolysis
- Glucose uptake in the muscle is inhibited
- Epinephrine inhibiting release of insulin from the pancreas to lessen any sort of rise in the serum insulin.

The second principal endocrine mechanism of maintaining or increasing blood sugars is through dynamizing pituitary adrenocortical axis, clinical studies are not clear in delineating how much or what type of stress gives this corticoid response.

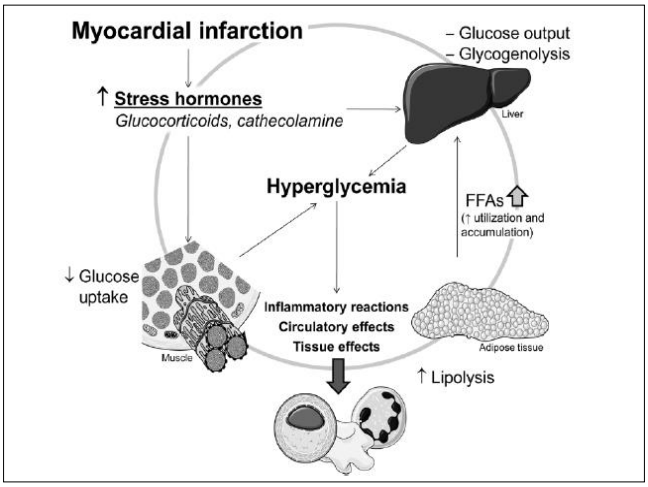

Figure 1: Cardiovascular effects of hyperglycemia during the acute phase of myocardial infarction.[5]

Relative insulin deficiency[6,7]

The effect counter regulatory hormones such as adrenaline, cortisol,glucagon and growth factors on the pancreas and peripheral cells is thought to cause relative insulin deficiency. They create a state of insulin resistance by decreasing insulin secretion.

Impaired glucose tolerance[7]

IGT not only important in developing overt diabetes and its associated complications, but also have an expanded risk of cardiovascular morbidity and mortality compared with patients with normal glucose tolerance.

Undiagnosed diabetes mellitus[7]

This forms a considerable subset of patients whose diabetic status is detected for the first time after an acute myocardial infarction insult. The true prevalence of diabetes mellitus among people with myocardial infarction might be as high as 45%, since diabetes is present in about 20% of individuals in an unselected population subclinically. There is an independent association between diagnosed and undiagnosed diabetes and increased mortality. Consequently it is of paramount importance to screen for diabetes in all patients admitted with chest pain as a common symptom.

Effects of hyperglycemia in acute myocardial infarction

The mechanisms underlying the detrimental association between dysglycemia and acute MI are not fully understood, but multiple hypotheses have been proposed.

Endothelial dysfunction

Damage to the endothelium plays an important role in the development and progression of atherosclerosis (Figure 2).

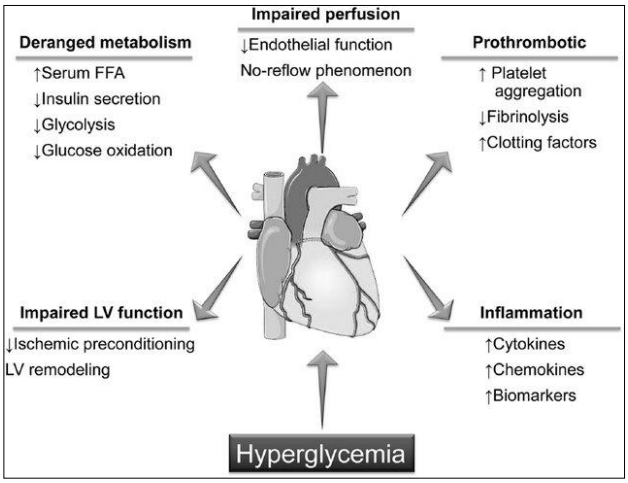

Figure 2: Cardiac effects of hyperglycemia during the acute phase of myocardial infarction.

Reduced collateral coronary[8,9]

Due to eNOS dysfunction there is decrease in arteriolar dilatation which obscures the normal increased flow and shear stress responsive element in the collateral vessel which is undergoing remodeling collaterization, as well as decrease endothelial cell permeability blood flow.

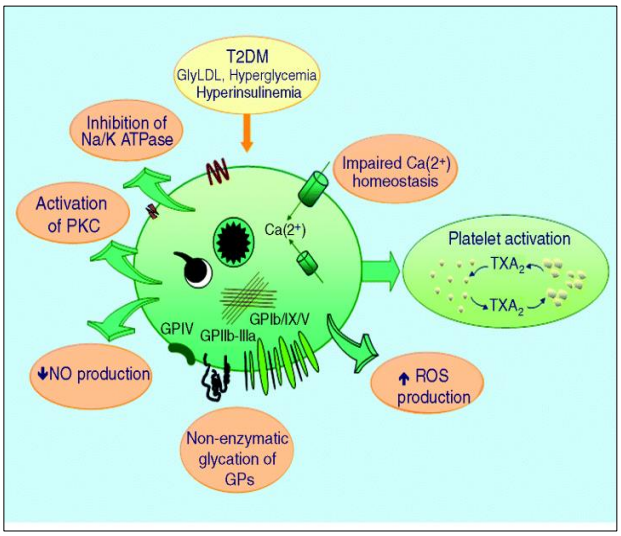

Figure 3: Impact of hyperglycemia on platelet function.

Increased thrombus formation

The surge in platelet adhesion and aggregation causes platelet dependent thrombin generation while decreasing vasodilatation mediated by platelets. Coagulation factors including von willebrand factor, factor VII, factor VIII and fibrinogen are significantly enhanced in a setting of hyperglycemia (Figure 3).

Amplification of inflammatory immune reaction

Numerous unpropitious effects are associated with acute hyperglycemia contributing to poor outcomes in acute coronary syndromes (ACS): promotion of inflammatory processes (including endothelial dysfunction, thrombosis, and platelet reactivity), metabolic derangements, increasing generation of free fatty acids and susceptibility to myocardial ischemia, and lower myocardial performance. Hyperglycemia is also a major predictor of left ventricular remodeling after ACS.[12]

Hyperglycemia promotes changes in the structure and conformation of platelets, as well as alterations of membrane lipid dynamics. Increased oxidative stress associated with hyperglycemia is responsible for activation of transcription factors and expression of redox-sensitive genes leading to a phenotypic switch of endothelium toward an adhesive, prothrombotic condition, initial platelet activation, adhesion, and subsequent platelet aggregate formation. There is also evidence that the prothrombotic state generate by hyperglycemia originates from reduced plasma fibrinolytic activity and action of tissue plasminogen activator. (GlyLD, glycated low-density lipoproteins; GP, glycoprotein; NO, nitric oxide; PKC, protein kinase C; ROS, reactive oxygen species).[13]

Table 1: Risk factors associated with the development of CAD.[14]

Non-Modifiable	Modifiable	New risk factors
Age	Hypertension	Atherogenic risk factors: Lipoprotein(a) Elevated Homocysteine level,
Presence of coronary heart disease	Dyslipidemia	
	Diabetes	
	Smoking	
Male gender	Diet	Plasma fibrinogen, Tissue plasminogen activator, C-Reactive protein.
Family history of CHD		
Menopause		
Physical inactivity		

Glycosylated haemoglobin[15,16]

The glycosylation of haemoglobin A to structure into HbA1c occurs all through the lifecycle of the erythrocyte, but occur faster in normal donor red cells given to diabetic recipients, the metabolic conformations in the diabetic patient accomplish glycosylation within red cells

circulating in their blood faster than occurs when the transfused red cells circulate in a normal recipient.

The level of glycosylated haemoglobin appears to be a reflection of blood sugars for a period of several weeks prior to the time of sampling.

It has therefore been suggested that the measurement of haemoglobin glycosylation would be a more stable indicator of the adequacy of control of diabetic state than occasional measurement of blood and urine glucose.

Formation of glycosylated haemoglobin[17]

Glucose reacts nonenzymatically with the NH2 terminal aminoacid of the beta chain of the human haemoglobin by way of keto amine linkage, resulting in the formation of glycosylated haemoglobin. The enhanced electrophoretic mobility of this fast moving minor haemoglobin component is due to the nonenzymatic glycosylation of the aminoacid valine and lysine.

Table 2: Conditions leading to falsely abnormal values for the hba1c.[18]

Factors influencing hemoglobin a1c		
Comorbidity	**Effect on RBC's**	**Effect on HbA1C**
Iron deficiency	RBC production decreases	Elevation
Vitamin B12 deficiency		
Lack of erythropoietin		
Pregnancy		
Renal failure		
Hemoglobinopathies	RBC destruction increases	Decline
Rheumatoid arthritis		
Spleenomegaly	RBC production increases	Decline
Elevated erythropoietin		
Chronic liver disease		
Splenectomy	RBC destruction decreases	Elevation

METHODS

Patient characteristics

Consecutive patients admitted to R. L. Jalappa hospital associated with Sri Devaraj Urs Medical College, and Narayana Hrudalaya, Kolar, Karnataka; for suspected ACS from November 2016 to October 2017, were eligible in this case study. All hospitalized patients were screened based on the admission diagnosis.

The whole spectrum of ACS, including unstable angina, non–ST-segment elevation myocardial infarction (NSTEMI), and STsegment elevation MI (STEMI), was studied.

The diagnosis of ACS was based on American College of Cardiology (ACC)/American Heart Association (AHA) guidelines. Patients with diabetes mellitus, chronic kidney disease, haemoglobinopathies and sepsis were excluded from the study.

Analysis of HbA1c on admission was done in every selected patient. Patients having HbA1c >6.5% were excluded from the study as they belonged to the diabetic category according to latest ADA guidelines.[19]

Table 3: Parameters.

Parameter	Study group (prediabetes)	Control group (non-diabetic)
Fasting plasma glucose	100-125mg/dl	<100mg/dl
Post-prandial glucose	141-199mg/dl	<140mg/dl
HbA1C	5.7-6.4%	<5.7%

Data collection

Data collection was done in a case record format. Demographic data and past medical history, including cardiovascular (CV) risk factors and comorbidities, were collected.

The investigation results including blood tests and electrocardiographic findings were also recorded. All patients were followed up till discharge.

Endpoints

The composite primary endpoints of this study were the correlation of HbA1c level with major adverse cardiac events (MACE) during hospital stay. MACE included CV mortality, arrhythmia, cardiogenic shock, congestive heart failure.

Statistical analysis

Authors used SPSS version 22 for statistical analysis. For the purpose of present analysis, patients were divided into 2 groups based on admission HbA1c: group 1, HbA1c ≥5.7% (the prediabetic group) and group 2, HbA1c< 5.7% (normal HbA1c group).

Quantitative data was presented as mean, standard deviation. ANOVA was the test of significance for quantitative data and chi-square test for the test of significance for qualitative data. A p value <0.05 was taken as statistically significant.

Association of various risk factors with MACE were analysed and significant variables were entered in a multivariate logistic regression analysis to determine independent predictability of risk factors.

RESULTS

Age distribution

Among cases majority 32.4% were in the age group 51 to 60 years and among controls majority 41.2% were in the age group 41 to 50 years. There was no significant difference in age distribution between two groups (Table 4).

Gender distribution

In cases, 61.8% were males and 38.2% were females and in controls 70.6% were males and 29.4% were females. There was no significant difference in gender distribution between two groups (Table 5).

Difference in co-morbidities

Among cases, 67.6% had HTN, 61.8% were smokers, 29.4% were alcoholics. Among controls, 61.8% had HTN, 44.1% were smokers, 5.9% had family history of CAD and 5.9% were alcoholics.

There was significant difference in alcohol consumption between cases and controls (Table 6).

Comparison of glycemic parameters

In the study there was significant difference in mean FBS, PPBS and HbA1c between cases and controls. All the three glycemic profile parameters were significantly higher in Cases than in controls. There was no significant difference in mean RBS between two groups (Table 7).

Association between hba1c, lipid profile with mace among cases and controls

Among cases there was significant association between Total Cholesterol, Triglycerides and LDL with mace (Table 8). Among controls, there was no significant association between Total Cholesterol, Triglycerides and LDL with mace (Table 9).

Table 4: Comparision of age distribution between two groups.

		Group					
		Cases		Controls		Total	
		Count	Percentage	Count	Percentage	Count	Percentage
Age	<40 years	7	20.6%	5	14.7%	12	17.6%
	41 to 50 years	10	29.4%	14	41.2%	24	35.3%
	51 to 60 years	11	32.4%	8	23.5%	19	27.9%
	>60 years	6	17.6%	7	20.6%	13	19.1%
	Total	34	100.0%	34	100.0%	68	100.0%

$\chi 2 = 1.551$, df =3, p =0.671

Table 5: Comparison of gender distribution between two groups.

		Group					
		Cases		Controls		Total	
		Count	Percentage	Count	Percentage	Count	Percentage
Gender	Female	13	38.2%	10	29.4%	23	33.8%
	Male	21	61.8%	24	70.6%	45	66.2%
	Total	34	100.0%	34	100.0%	68	100.0%

Table 6: Comorbidities and past history distribution comparison between two groups.

		Group						p value
		Cases		Controls		Total		
		Count	Percentage	Count	Percentage	Count	Percentage	
Hypertension	No	11	32.4%	13	38.2%	24	35.3%	0.612
	Yes	23	67.6%	21	61.8%	44	64.7%	
Smoker	No	13	38.2%	19	55.9%	32	47.1%	0.145
	Yes	21	61.8%	15	44.1%	36	52.9%	
Family history of CAD	No	34	100.0%	32	94.1%	66	97.1%	0.151
	Yes	0	0.0%	2	5.9%	2	2.9%	
Alcohol	No	24	70.6%	32	94.1%	56	82.4%	0.011*
	Yes	10	29.4%	2	5.9%	12	17.6%	

Table 7: Comparision of RBS, FBS, PPBS and HbA1C between two groups.

	Group						p value
	Cases		Controls		Total		
	Mean	SD	Mean	SD	Mean	SD	
RBS at admission	168.59	77.53	141.74	19.90	155.16	57.78	0.055
FBS	117.09	6.18	92.59	11.16	104.84	15.25	<0.001*
PPBS	164.00	18.85	131.65	12.74	147.82	22.81	<0.001*
HbA1c	6.09	0.27	5.32	0.30	5.70	0.48	<0.001*

Table 8: Association between HBA1C, lipid profile with mace among cases.

		Mace								p value
		Cardiogenic shock		Congestive heart failure		No complications		Ventricular tachycardia		
		Count	Percentage	Count	Percentage	Count	Percentage	Count	Percentage	
Total cholesterol	>200 mg/dl	8	33.3%	5	20.8%	3	12.5%	8	33.3%	0.044*
	<200 mg/dl	0	0.0%	1	10.0%	5	50.0%	4	40.0%	
Triglycerides	>150 mg/dl	8	28.6%	6	21.4%	3	10.7%	11	39.3%	0.002*
	<150 mg/dl	0	0.0%	0	0.0%	5	83.3%	1	16.7%	
LDL	>129 mg/dl	8	25.8%	6	19.4%	5	16.1%	12	38.7%	0.014*
	<129 mg/dl	0	0.0%	0	0.0%	3	100.0%	0	0.0%	

Group = Cases

Table 9: Association between HBA1C, lipid profile with mace among controls.

		Mace								p value
		Cardiogenic shock		Congestive heart failure		No complications		Ventricular tachycardia		
		Count	Percentage	Count	Percentage	Count	Percentage	Count	Percentage	
Total cholesterol	>200 mg/dl	1	20.0%	2	40.0%	2	40.0%	0	0.0%	0.217
	<200 mg/dl	2	6.9%	3	10.3%	23	79.3%	1	3.4%	
Triglycerides	>150 mg/dl	1	4.5%	3	13.6%	17	77.3%	1	4.5%	0.571
	<150 mg/dl	2	16.7%	2	16.7%	8	66.7%	0	0.0%	
LDL	>129 mg/dl	2	8.7%	5	21.7%	15	65.2%	1	4.3%	0.316
	<129 mg/dl	1	9.1%	0	0.0%	10	90.9%	0	0.0%	

Group = Controls

Table 10: Diagnosis comparison between two groups.

		Group					
		Cases		Controls		Total	
		Count	Percentage	Count	Percentage	Count	Percentage
Diagnosis	NSTEMI	14	41.2%	16	47.1%	30	44.1%
	STEMI	14	41.2%	10	29.4%	24	35.3%
	Unstable angina	6	17.6%	8	23.5%	14	20.6%
	Total	34	100.0%	34	100.0%	68	100.0%

$\chi 2$ =1.086, df =2, p =0.581

Comparison of diagnosis

Among cases, 41.2% had NSTEMI, 41.2% had STEMI and 17.6% had Unstable Angina and among controls, 47.1% had NSTEMI, 29.4% had STEMI and 23.5% had unstable angina. There was no significant difference in diagnosis between two groups (Table 10).

2D echo comparison between two groups

Among cases,

- 14.7% had Normal LV Function
- 29.4% had Mild LV Dysfunction
- 44.1% had Moderate LV Dysfunction
- 11.8% had Severe LV Dysfunction.

Among controls,

- 26.5% had Normal LV Function
- 52.9% had Mild LV Dysfunction
- 11.8% had Moderate LV Dysfunction
- 8.8% had Severe LV Dysfunction.
- There was significant difference in 2D Echo findings between two groups (Table 11).

Mace and HbA1C

In this study among those with HbA1c >5.7, 21.9% had no complications, 25% had Cardiogenic shock, 18.8% had CCF and 34.4% had Ventricular Tachycardia. Among those with HbA1c <5.7, 72.2% had No complications, 8.3% had Cardiogenic Shock, 13.9% had Congestive Heart Failure and 5.6% had Ventricular Tachycardia. There was significant association between HbA1c and MACE (Table 12).

Table 11: 2D echo comparison between two groups.

		Group					
		Cases		Controls		Total	
		Count	Percentage	Count	Percentage	Count	Percentage
2D Echo	Normal LV function	5	14.7%	9	26.5%	14	20.6%
	Mild LV dysfunction	10	29.4%	18	52.9%	28	41.2%
	Moderate LV dysfunction	15	44.1%	4	11.8%	19	27.9%
	Severe LV dysfunction	4	11.8%	3	8.8%	7	10.3%
	Total	34	100.0%	34	100.0%	68	100.0%

$\chi 2$ =9.940, df =3, p =0.019*

Table 12: Association between mace and HBA1C.

		Mace							
		No complications		Cardiogenic shock		Congestive heart failure		Ventricular tachycardia	
		Count	Percentage	Count	Percentage	Count	Percentage	Count	Percentage
HbA1c	>5.7	7	21.9%	8	25.0%	6	18.8%	11	34.4%
	<5.7	26	72.2%	3	8.3%	5	13.9%	2	5.6%

$\chi 2$ =19.366, df =3, p <0.001*

DISCUSSION

Age and gender between prediabetic and non diabetics

In our study the mean age in prediabetic ACS patient was 51 to 60 years and that of non diabetic 41 to 50 years indicating the absence of a statistically significant difference between age of diabetic patients when compared to non diabetic patients.

In cases, 21 were male patients and 13 were female patients. Among controls 24 were male patients and 10 were female patients. The male and female comparison between the two groups was not statistically significant (p=0.442).

There was no gender and age preponderance between the prediabetics and non diabetics (Table 4 and Table 5).

Mode of presentation in ACS

In our study, among cases, 41.2% had STEMI, 41.2% had NSTEMI and 17.6% had Unstable Angina and among controls, 47.1% had NSTEMI, 29.4% had STEMI and 23.5% had Unstable angina. There was no significant difference in mode of presentation between two groups (Table 10).

ACS and clinical findings

In our study, among cases 23 patients had hypertension, 21 patients were smokers and 10 patients were alcoholic. Among controls, 44 patients had hypertension, 36patients were smokers, 2 patients had family history of coronary artery disease and 12 were alcoholics. There was no statistically significant difference between number of smokers and prevalence of hypertension between the groups. There was significant difference in Alcohol consumption between cases and controls (Table 6).

Similar observations were noted in several other studies which have proven that hypertension and alcohol consumption were common co-morbidities.[20]

ACS and clinical outcome

Our study showed that 41.2% had ST Elevation MI, 41.2% had Non ST Elevation MI and 17.6% had Unstable Angina. While population based studies have shown that up to 23.1% of patients presented with ACS has ST elevation Ml.

In our patients, HbA1c >5.7, 25% had Cardiogenic shock, 18.8% had CCF and 34.4% had Ventricular Tachycardia. In this study, the most common adverse cardiac event observed was ventricular tachycardia (Table 9) Study by Vinita Elizabeth Mani and John, in which 47.1% patients having arrhythmia were in low HbA1C group and 52.9% patients having arrhythmia were in high HbA1C group also support this study.[21] In our study, authors found that most of the patients with HbA1C>5.7% had lower EF i.e. 29.4% had Mild LV Dysfunction, 44.1% had Moderate LV Dysfunction and 11.8% had Severe LV Dysfunction as compared to patients with HbA1C<5.7%, who had higher LVEF (Table 8).

A study done by Razzaq et al, demonstrated that the mean EF was significantly lower in a group of HbA1C 6.5-8.5 and in HbA1C> 8.5 as contrasted with that group <6.5.[22] A linear decline in EF was seen with increasing HbA1C level in patients with ACS. 16 out of 100 patients had heart failure. 11 patients belong to high normal HbA1C and 5 belong to normal HbA1C group. This is supported by the study given below. A study by John and Mani, 27% patients of heart failure were in low HbA1C group(<7%) and 73% patients with heart failure were in high HbA1C group(>7%).[21] In our study 18.8% patients of heartfailure were in high HbA1C(>5.7%) and 13.9% patients of heart failure were in low HbA1C group(<5.7%). These findings suggests that as there is rise in HbA1C value the chance of heart failure rises.

Blood sugars, HbA1c and clinical outcome

The knowledge of correlation of FBS and PPBS with HbA1c may be helpful in the management of cases to achieve good glycemic control. The exact contributions of PPBS and FBS to overall glycaemia remain controversial. There is limited evidence to suggest which one among the FPG and PPBS glucose is the dominant contributor to overall glycaemia in patients with T2DM. In our study there was significant difference in mean FBS, PPBS and HbA1c between cases and controls. All the three glycemic profile parameters were significantly higher in Cases than in controls. There was no significant difference in mean RBS between two groups (Table 7). Similar observation was made in various other studies.[27]

Elevated plasma sugar value in subjects hospitalised for MI seems to be a frequent phenomenon. Studies have pointed out that there is a greater rate of mortality and other complications due to this elevation in both group of individuals with and without DM.[27] The correlation between enhanced plasma glucose on hospitalization and adverse consequences might be the parameter which is independent of other prognostic determinants.

Lipid profile and MACE

In a study done by Rahbar et al showed that pre-diabetics are at higher risk of having low level of HDL cholesterol (HDL-c).[23] Impaired lipid profile i.e. dyslipidemia associated with CVD in type 2 diabetes and can also occur in pre-diabetics.

In our study, subjects with HbA1C levels >5.7, 70.6% had Total cholesterol >200 mg/dl and 91.2% had LDL >129 mg/dl and had higher chances of MACE probably attributing to acceleration of macrovascular atherosclerosis (Table 8, Table 9).

A study carried out by Gaziano et al and Boizel et al showed that TG/HDL were significantly higher in IFG/ IGT compared to NFG/NGT.[24,25] The same was observed in a study conducted by Miyazaki et al that IFG/IGT subjects had higher TG/ HDL ratio (4.0±2.5 for cases and 2.7±1.9 for controls).[26] These results suggested that elevation of postprandial levels of plasma glucose and insulin based on whole body insulin resistance contributed to atherogenic lipids profile. This study was limited with respect to population size and the patients were followed only till the time of discharge. This leaves us blind about the long term complications which could be effected by HbA1C.

With this study, a scope for further investigation regarding long term complications and complications associated with fluctuating levels of blood sugars may be considered. Large sample size is required to confirm the age, and gender difference in ACS outcome.

CONCLUSION

This study showed that HbA1C is a significant predictor of MACEs after AMI in prediabetic patients. This biomarker may strengthen the accuracy of clinical care in early intervention and secondary prevention. HbA1C may be considered as effective indicator that facilitates the

early detection of patients with potential adverse prognosis.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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