ASIAN JOURNAL OF PHARMACEUTICAL AND CLINICAL RESEARCH

INNOVARE ACADEMIC SCIENCES Knowledge to Innovation

Vol 7, Issue2, 2014

ISSN - 0974-2441

ResearchArticle

CORRELATION BETWEEN GLYCEMIC CONTROL AND LIPID PROFILE IN TYPE 2 DIABETIC PATIENTS: HbA1c AS AN INDIRECT INDICATOR OF DYSLIPIDEMIA

SREENIVAS REDDY A [1]*, MEERA S [2], EBENEZER WILLIAM [3], KUMAR J S.[4]

[1]Tutor, [2] Associate Professor, [3] Professor & Head Department of Biochemistry [4] Professor Department of General Medicine &Diabetologe SRM Medical College, Hospital & Research Centre, Kattankulathur, Tamil Nadu, India. Email: sri_biochemistry@yahoo.com

Received: 25 January 2014, Revised and Accepted:22 February 2014

ABSTRACT

Objectives: Dyslipidemia is one of the major risk factor for cardiovascular disease in Type 2 Diabetes mellitus, characterized by elevated Total cholesterol (TC), Triglycerides (TG), Low density lipoprotein (LDL) and decreased High density lipoprotein (HDL). Hemoglobin A1c (HbA1c) is widely used as an index of mean glycaemia, a measure of risk for the development of diabetes complications and a measure of the quality of diabetes care. The aim of this study was to determine the impact of glycemic control on lipid profile and to know utility of HbA1c as an indirect indicator of dyslipidemia.

Methods: A total of 490 Type 2 Diabetes mellitus patients (males 258, females 232) mean age 53.17 years standard deviation (S.D) 10.50 were included in this study.

Results: The age of type 2 diabetic patients were not significantly correlated with fasting blood glucose and HbA1c. The level of HbA1c was highly direct significant correlation with fasting blood glucose. The Age of Type 2 Diabetic patients were highly significant and inversely correlated with Total cholesterol, Triglycerides, Low-density Lipoprotein, where it was not significantly correlated with High-density Lipoprotein. The level of HbA1c was highly direct significant correlation with TC, TG, LDL, where it was not correlated with HDL.

Conclusion: The findings of this study clearly showed that HbA1c is not only a reliable glycemic index but also as an indirect indicator of dyslipidemia.

Keywords: Type 2 diabetes mellitus, HbA1c, Lipid profile, Dyslipidemia.

INTRODUCTION

Type 2 diabetes mellitus is a group of metabolic disorder that is characterized by hyperglycemia resulting from insulin resistance and relative insulin deficiency [1]. Diabetes is associated with a greater risk of morbidity and mortality from cardiovascular disease (CVD). Serum lipids are frequently abnormal and are likely to contribute to the risk of coronary artery disease [2]. Worsening of glycemic control deteriorates lipid and lipoprotein abnormalities and particularly of diabetes mellitus [3]. The American Diabetes study (ADA) has designated HbA1c level of <7% as a goal of optimal blood glucose control [4] and the American Association of Clinical Endocrinologist has further recommended HbA1c level of <6.5% [5]. Criteria for abnormal lipid profiles were based on the ADA criteria, Hypercholesterolemia refers to a total cholesterol level ≥ 200 mg/dl, Hypertriglyceridemia refers to a level is ≥ 150 mg/dl, HDL was considered low when the level is < 40 mg/dl in males and < 50 mg/dl in females, LDL was considered high when the level is ≥ 100 mg/dl. Dyslipidemia was defined as the presence of one or more of the previous abnormalities in serum lipids [6]. HbA1c is formed by the condensation of glucose with the N-terminal Valine residue of each β-chain of HbA to form an unstable Schiff-base, which is the most widely used biomarker for long-term glycemic status, as well as an independent risk factor for coronary heart disease (CHD) and stroke [7]. Elevated cholesterol levels, are believed to be a major factor in promoting atherosclerosis, it is now recognized that triglycerides are an independent risk factor. Atherosclerosis is characterized by the deposition of cholesterol and cholesterol from the plasma lipoproteins into the artery wall. Diseases in which prolonged elevated levels of VLDL, IDL, chylomicron remnants, or LDL occur in the blood (diabetes mellitus). The liver and many extra-hepatic tissuesexpress the LDL (apo B-100) receptor. It is useful for atherogenic risk assessment in dysglycemic patients (8). HDL is synthesized and secreted from both liver and intestine. However ,apo C and apo E are synthesized in the liver and transferred from liver HDL to intestine HDL, when the latter enters the plasma. Non-HDL is a simple, readily , no-cost test obtained with the usual lipid profile and reflects the atherogenic risk in diabetic patients with hypertriglyceridemia and can conveniently measure CVD risk (9). The aim of this study was to examine impact of glycemic control on the lipid profile of type 2 diabetic patients, and to know importance of HbA1c as an indirect indicator of dyslipidemia.

MATERIAL AND METHODS

A comparative and cross sectional study was conducted among 490 type 2 diabetic patients. Institutional Ethical Committee approved the study and informed consent was obtained from the patients. HbA1c assay was done by high performance liquid chromatography (HPLC). Fasting Blood Glucose, Total cholesterol, Triglycerides and HDL was measured by enzymatic method by using OLYMPUS AU-400 Auto-analyzer on the same day of collection. The levels of LDL by using Friedewald's formula.

Inclusion criteria

Type 2 Diabetic patients aged 30 to 75 years of both male and female who visited SRM Medical College, Hospital & Research Centre were included in this study.

Exclusion criteria

Type 2 Diabetic patients with liver disease, thyroid disorders, terminally illness were excluded.

Statistics

The results were evaluated by SPSS statistical package version 20 by one-way analysis of variance (ANOVA) followed by Bonferroni multiple comparison test and students t test. The results were expressed as Mean ± Standard deviation (S.D); $P < 0.05$ was

considered statistically significant. Values of HbA1c % and FBS, lipid profile were given in mg/dl.

RESULTS

Table 1 shows the mean and standard deviation of all type 2 diabetic patients age (53.17±10.50), FBG (171.37 ± 75.66), HbA1c (8.36 ± 2.20), TC (190.08 ± 45.77), TG (156.70 ± 110.51), HDL (44.30 ± 10.79) and LDL (114.65 ± 38.36). The age of patients did not show any significant correlation with either FBG (Pearson correlation -.82, P=0.69 or HbA1c (Pearson correlation -.65, P=0.15, Table 2). Although there was no significant correlation between age and HDL (Pearson correlation .060, p=0.18, Table 4). The age of the patients significantly and inversely correlated with Total Cholesterol (Pearson Correlation -.197, P=0.000), Triglycerides (Pearson Correlation -.102, P=0.02) and Low-density Lipoprotein (Pearson Correlation -.202, P=0.000, Table 4). The levels of FBG highly direct correlation with Total Cholesterol (Pearson Correlation .242, P=0.000), Triglycerides (Pearson Correlation .171, P=0.000) and LDL (Pearson Correlation .201, P=0.000) where it was not correlated with HDL (Pearson Correlation -.022, P=0.62, Table 4). HbA1c was highly direct correlation with Fasting Blood Glucose (Pearson Correlation .773, P=0.000, Table 3), TC (Pearson Correlation .227, P=0.000), TG (Pearson Correlation .155, P=0.001), LDL (Pearson Correlation .187, P=0.000) where it was not correlated with HDL (Pearson Correlation -.005, P=0.90, Table 4).

The impact of glycemic control on lipid profile was evaluated by categorizing all the patients into three groups on the basis of HbA1c levels. Group A consists of patients with HbA1c value 4.5-6.5%, Group B consists of patients with HbA1c value 6.6-8.5%, Group C consists of patients with HbA1c value 8.6-16%. Out of 490 Type 2 diabetic patients 258 were males and 232 were females. The age (ANOVA) F=1.5, P=0.22 and HDL (ANOVA) F=1.65, P=0.19, were not significantly correlated in group C and group B than in group A (Table 5). The concentration of FBG (ANOVA) F=204.68, P=0.22, HbA1c (ANOVA) F=719.22, P=0.000, TC (ANOVA) F=9.82, P=0.000, TG (ANOVA) F=8.19, P=0.000, LDL (ANOVA) F=5.54, P=0.004 were highly significant differences in group C and group B than in group A.

Table 1: Mean ± S.D values of Type 2 Diabetic patients.

Parameter	Total of all type 2 diabetic patients (N= 490) Male 258, Female 232	
	Mean	**Standard Deviation (S.D)**
Age	53.17	10.50
FBG	171.37	75.66
HbA1c	8.36	2.20
TC	190.08	45.77
TG	156.70	110.51
HDL	44.30	10.79
LDL	114.65	38.36

Table 2: Correlation between Age and FBG, HbA1c in all Type 2 Diabetic subjects

Parameter	FBG		HbA1c	
	P.C	P	P.C	P
Age	-0.082	0.69 N.S	-0.065	0.15 N.S

P.C →Pearson's Correlation, N.S. Not Significant

Table 3: Correlation between HbA1c and Fasting blood glucose in type 2 Type Diabetic subjects

Parameter	FBG	
	Pearson Correlation	P-Value
HbA1c	0.773	0.000***

***** Highly Significant**

Table 4: Correlation between Lipid Profile and Age, FBG, HbA1c in all Type 2 Diabetic subjects

Parameter	Age		FBG		HbA1c	
	P.C	P	P.C	P	P.C	P
TC	-0.197	0.000***	0.241	0.000***	0.227	0.000***
TG	-0.102	0.02***	0.171	0.000***	0.155	0.001***
HDL	0.06	0.18 N.S	-0.022	0.62 N.S	-0.005	0.90 N.S
LDL	-0.202	0.000***	0.201	0.000***	0.187	0.000***

P.C → Pearson's Correlation, * Highly Significant, N.S. Not Significant**

Table 5: Comparison of Mean, S.D, F and P values of measured parameters between good, poor and worst glycemic control.

Parameter	Group A	Group B	Group C	F-Value	P-Value
Age	53.76 ± 10.79	53.87 ± 10.94	52.14 ± 10.19	1.5	0.22 N.S
FBG	114.88 ± 20.66	143.63 ± 30.82	233.76 ± 82.85	204.68	0.000***
HbA1c	6.09 ± 0.42	7.49 ± 0.56	10.64 ± 1.66	719.22	0.000***
TC	179.28 ± 42.92	186.05 ± 43.09	200.87 ± 47.86	9.82	0.000***
TG	130.61 ± 70.29	150.26 ± 85.63	179.74 ± 143.26	8.19	0.000***
HDL	45.74 ± 10.78	43.49 ± 9.09	44.09 ± 12.09	1.65	0.19 NS
LDL	107.41 ± 35.53	112.50 ± 38.01	121.37 ± 39.55	5.54	0.004***

***** Highly Significant, N.S. Not Significant**

DISCUSSION

In our study, the usual Lipid profile, fasting blood glucose and Hemoglobin A1c was investigated. Abnormality of Cholesterol metabolism may lead to cardiovascular disease and heart attacks. Total cholesterol levels are different in the presence of risk factor for diabetes mellitus. The National Cholesterol Educational Program (NCEP) identified elevated LDL as a primary risk factor for coronary heart disease (CHD) [10]. This study reveals high prevalence of hypercholesterolemia, hypertriglyceridemia, high LDL and low HDL levels which are well known risk factors for cardiovascular disease. Insulin affects the liver Apo-lipoprotein production. It regulates the enzymatic activity of lipoprotein lipase (LpL) and cholesterol ester transfer protein (CETP). All these factors are likely cause of dyslipidemia in diabetes mellitus [11]. The significant correlation between Hemoglobin A1c and Fasting Blood Glucose (Table 3) are in accordance with previous study [12, 13]. Alternations in serumbiochemical parameters in old age groups of patients had significantly lower TC, TG and LDL with earlier reports [14].Our diabetic study reveals age was not interpreted to HbA1c and FBG, as well as age was significantly inverse correlation with TC, TG, and LDL. But HbA1c was highly direct significant correlation with TC, TG and LDL. Worse glycemic control had significantly high TC, TG and LDL levels, but not in HDL levels.

CONCLUSION

The glycemic control of the patient has got a strong impact on the serum lipid profile levels and atherosclerosis, CVD and CHD including heart attack and stroke. Patients should be educated about regular monitoring of lipid profiles and if found to be abnormal, should control blood glucose and cholesterol very effectively. Thus our diabetic study clearly added value of HbA1c can be monitoring

long-term glycemic control and as an indirect indicator of dyslipidemia in type 2 diabetic patients.

ACKNOWLEDGMENT

I would like to thank the department of general medicine and Clinical Biochemistry Laboratory at SRM Medical College, Hospital & Research Centre for valuable help

REFERENCES

1. Taskinen MR (2003) Diabetic dyslipidemia: from basic research to clinical practice. Diabetologia 46; 733-749.
2. Jamshaid T, Qureshi A. Hyperlipidemia in Diabetics. Pac Postgrad Med J 2002; 13: 159-60.
3. Grundy SM. Hypertriglyceridemia, insulin resistance, and the metabolic syndrome. Am J Cardiol 2006; 83: 25-29.
4. American Diabetes Association. (2003). Implications of the United Kingdom Prospective Diabetes study. Diabetes Care, 26, 28-32.
5. The American Association of Clinical Endocrinologists medical guidelines for the management of diabetes mellitus. (2002. The AACE system of intensive diabetes self-maagement-2002 update. Endocrine Pracice, 8, 40-82.
6. America Diabetes Association (2004). Dyslipidemia management in adult with diabetes. Diabetes care, 24,68-71.
7. Selvin E, Coresh J, Shahar E et al (2005) Glycaemia (hemoglobin A1c) and incident of ischemic stroke: the Atherosclerosis Risk in Communties (ARIC) Study. Lancet Neurol 4:821-826.
8. Ponguzhali D, V. M.Vinodhini, Ebenezer William W, Kumar J S, Evaluation of apolipoprotein-B levels in dysglycemia. Asian J Pharm Clin Res 2013; 6(3): 112-114.
9. Arul Senghor, Ebenezer William, Non-HDL and AIP compared to Hs-CRP in hypertriglyceridemic diabetics – A better cardiovascular risk marker? Senghor et al. Asian Pharm Clin Res, Vol 6 issue 4 , 2013, 128-130
10. National Cholesterol Education Program (NCEP). Lipid Panel Reference Ranges: Pathology, inc 2011 Nov, 2.
11. Goldberg IJ. Lipoprotein lipase and lipolysis: central roles in lipoprotein metabolism and atherogenesis. J Lipid Res 1996; 37: 693-707
12. Ito. C. Maeda R. Ishida S. Sasaki H. Harada H. Correlation among fasting plasma glucose, two-hour plasma glucose levels in OGTT and HbA1c. Diabetes Res ClinPract 2000; 50: 225-230.
13. Rosediani M. Azidah AK. Mafauzy M Correlation between fasting plasma glucose, post prandial glucose and glycatedhemoglobin and fructosamine. Med J Malaysia 2006; 61: 67-71.
14. Walden C, Knopp R, Wahl P, Beach K, Strandness E (1984) Sex differences in the effect of diabetes mellitus on lipoprotein triglyceride and cholesterol concentrations. N Engl J Med 311:953-959.