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To Evaluate the Association of Serum Adiponectin with Fasting and Postprandial Plasma Glucose and Fasting Lipids in Control, IGT and T2DM Subjects

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Abstract **Original Research Article**

Background: Association of adiponectin with insulin resistance occurs early in obesity development; however it remains unclear if this association is of further importance in prediabetes and early stages of type 2 diabetes and remains the same through all glucose intolerance development stages. The present study was undertaken to evaluate the association of dysglycemia and dyslipidemia with adiponectin in prediabetic subjects as well as in T2DM subjects. ***Objective:*** The objective of our study was to evaluate adiponectin level in subjects with pre-diabetes and to compare it with the levels in newly diagnosed type 2 diabetes and healthy (normal glucose tolerance) subjects. ***Method:*** It was an observational analytic study with a group compare design. The study was conducted in the Biomedical Research Group and Department of Biochemistry & Cell Biology of the Bangladesh Institute of Research and Rehabilitation in Diabetes, Endocrine and Metabolic Disorders (BIRDEM), Dhaka. This study was done during the period of July 2012 to June 2013. ***Results:*** Bivariate Spearman's correlation analyses were performed for adiponectin with clinical, anthropometrical and other biochemical variables in the Control, IGT and T2DM groups. Postprandial glucose (p=0.023) and age (p=0.011) had a positive correlation with adiponectin in control subjects. A negative correlation was observed between adiponectin and BMI in IGT subjects (p=0.004). A positive correlation with adiponectin and serum HDL (p=0.03) and a negative correlation with serum LDL (p=0.03) were found in T2DM subjects. ***Conclusion:*** From our study we can conclude that, BMI has a negative association with adiponectin in IGT subjects. Serum LDL has a negative association and HDL has a positive association with adiponectin in IGT and T2DM subjects.

Keywords: Serum adiponectin, type 2 diabetes, postprandial glucose.

INTRODUCTION

The global prevalence of T2DM is expected to be double in the period 2000-2025 and may reach a level of almost 300 million people [1]. The progressive deterioration of pancreatic insulin secretion has been implicated as the proximate cause of the progressive increase in plasma glucose level [2]. Thus decrease in insulin secretion is a major contributor to the development of the overt T2DM state.

Plasma adiponectin levels do not decline with age. They are negatively associated with visceral and sub-cutaneous abdominal fat and plasma leptin level; and positively associated with glucose utilization across the adult age span [3]. Serum adiponectin concentrations were lower in the glucose tolerance

groups amongst women who later developed diabetes compared with those who did not. Low adiponectin concentrations were associated with future diabetes independently of insulin secretion and sensitivity, as well as IGT, IFG, smoking and abdominal obesity [4].

Some clinical studies on the regulation of adiponectin have been performed. The plasma adiponectin concentrations were inversely correlated with fasting glucose, fasting insulin concentrations, triglyceride, and body mass index, but positively correlated with HDL-cholesterol [5, 6]. Reduced serum levels of adiponectin in obese compared with nonobese subjects and negative correlations between adiponectin and body mass index (BMI) have been reported **[7,** 8**].** However, the relationship between BMI and circulating adiponectin becomes statistically insignificant if factors related to insulin sensitivity are taken into account suggesting that obesity does not influence serum adiponectin concentrations [9, 10]. Another study on twins, provide novel information on the relative effects of environmental and genetic factors on adiponectin and BMI, two quantitative traits that are relevant for IR, the metabolic syndrome, and type 2 diabetes mellitus. They indicate that both adiponectin and BMI are genetically determined, and that minor influences of environmental factors unique to individuals also exist [11].

OBJECTIVES

The objective of the study was to investigate the association of serum adiponectin with fasting and postprandial plasma glucose and fasting lipids in control, IGT and T2DM subjects

METHODOLOGY

Type of study	It was an observational analytic study with a group compare design.
Place of study	The study was conducted in the Biomedical Research Group and Department of Biochemistry & Cell Biology of the Bangladesh Institute of Research and Rehabilitation in Diabetes, Endocrine and Metabolic Disorders (BIRDEM), Dhaka.
Study period	This study was done during the period of July 2012 to June 2013.
Study population	A group of 71 subjects were included in this study and they were recruited from the Out-Patient Department of the BIRDEM Hospital.
Sampling technique	Purposive

Inclusion Criteria

- Adult subjects with age ranging from 22-57 years.
- Voluntarily agreed to include in this study by providing informed consent.

Exclusion Criteria

- Patients with serious co-morbid diseases (severe infection, stroke, myocardial infarction, major surgery, mal-absorption etc).
- History of using drugs significantly affecting glucose metabolism (glucocorticoids, oral contraceptives containing levonorgestrel or high-dose estrogen, phenytoin, high-dose thiazide diuretics etc.)
- Pregnancy.

Statistical Analysis

Data were expressed as mean ±SD and/or median (range) where appropriate. Comparison between two groups was done using Students 't' test (paired and unpaired), Mann-Whitney 'U' test and Wilcoxon 'Z' test. Bivariatte correlation analysis was done by using Spearman's Correlation analysis. To adjust the effects of confounder variables multiple linear regression analysis was done taking serum adiponectin level and fasting glucose as a dependent variable. Data were managed and statistical analyses were performed using Statistical Package for Social Science (SPSS) for Windows version 11.5. A p value <0.05 was taken as level of significance.

RESULT

Clinical and anthropometric characteristics of the study subjects

Table 1: Clinical and anthropometric characteristics of the study subjects

Variable	Control(22)	IGT(36)	T2DM(13)	t/p value		
				Control/IGT	Control/T2DM	IGT/T2DM
AGE(years)	38.75±8.40	42.71±7.64	44.61±7.00	1.774/ 0.076	-2.449/ 0.022	-0.727/ 0.473
BMI (kg/m^2)	20.53±2.43	20.89±2.22	21.14±2.23	0.556/ 0.581	-0.796/ 0.430	-0.322/ 0.749
WHR	0.905±0.03	0.927±0.03	0.922±0.02	2.465/ 0.019	-2.102 0.048	0.399/ 0.693
Creatinine(mg/dl)	0.936±0.16	0.957±0.17	0.981±0.17	0.459/ 0.648	0.787/ 0.436	0.394/ 0.696
SGPT(u/l)	22.22±8.99	26.00±19.57	27.00±15.74	0.987 /0.327	1.276 /0.208	-0.145/ 0.886

Results were expressed as Mean±SD. n=number of subjects; BMI, body mass index; WHR, waist to hip ratio; serum creatinine, serum SGPT: serum glutamate pyruvate kinase IGT, impaired glucose tolerance; T2DM, type 2 diabetes mellitus.

Table 1 shows that mean (±SD) age in the Control, IGT and T2DM subjects were 38.75±8.40, 42.71±7.65 and 44.61±7.00 years respectively. Mean (±SD) BMI in the Control, IGT and T2DM subjects were 20.53±2.43, 20.89±2.22 and 21.14±2.23 respectively. Mean BMI of IGT and T2DM did not show statistically significant difference compared to the Control (Table 1). Mean WHR in the IGT and T2DM group were statistically significant difference compared to the Control (p=0.019,p=0.048) but T2DM group did not show any statistically significant to IGT (Table 1). Mean (±SD) serum creatinine value of Control, IGT and subjects were 0.936±0.16,0.957±0.17 and 0.95±0.15 respectively. The value did not show statistically significant with each other. Mean (±SD) value of SGPT of Control, IGT and subjects were 22.22±8.99,26.00±19.57 and 27.00±15.74 respectively.

Correlation analyses

Spearman's correlation of serum adiponectin with different variables

Bivariate Spearman's correlation analyses were performed for adiponectin with clinical, anthropometrical and other biochemical variables in the Control, IGT and T2DM groups.

In control group, serum adiponectin did not show any significant correlation with BMI, WHR, fasting serum glucose, TG, LDL, Cholesterol, but showed significant positive correlation with Age, postprandial glucose, HDL (Table 2).

In IGT group, Serum adiponectin did not show any significant correlation with age, WHR, TG, fasting serum glucose, Postparendial glucose, HDL, LDL, but showed positive correlation with BMI (Table 2).

In T2DM group, serum adiponectin showed significant positive correlation with HDL, but showed negative correlation with LDL (Table 2).

Table 2: Spearman's correlation of serum adiponectin with various parameters among the Control Subjects

Group		Age	BMI	FBG	Postprandial	WHR	TG	Chol	HDL	LDL
Control(n=22)	r	0.379	0.105	-0.117	0.420	-0.097	-0.086	-0.134	-0.419	-0.003
	p	0.023	0.542	0.495	0.011	0.572	0.618	0.435	0.011	0.985

Results were expressed as Spearman's correlation coefficient r and statistical significance p. n=control. BMI, body mass index; WHR, waist-hip ratio; FBS, fasting serum glucose; 2h-AG, postprandial glucose; TG, triglyceride; HDL, high density lipoprotein. LDL, low density lipoprotein.

Table 3: Spearman's correlation of serum adiponectin with various variables among the IGT Study Subjects

Group		Age	BMI	FBG	Postprandial	WHR	TG	Chol	HDL	LDL
IGT(n=3)	r	0.095	0.606	-0.093	-0.346	0.298	-0.305	0.108	0.072	0.381
	p	0.683	0.004	0.688	0.124	0.190	0.179	0.641	0.758	0.089

Results were expressed as Spearman's correlation coefficient r and statistical significance p. IGT, impaired glucose tolerence; BMI, bodymass index; FBS, fasting serum glucose; TG, triglyceride; HDL, high density lipoprotein; LDL, low density lipoprotein.

Table 4: Spearman's correlation of serum adiponectin with various variables among the T2DM Study Subjects

Group		Age	BMI	FBG	Postprandial	WHR	TG	Chol	HDL	LDL	Creat	SGPT
T2DM(n=13)	r	0.183	-0.421	-0.207	0.017	0.229	0.149	0.506	0.324	-0.626	-0.099	0.082
	p	0.590	0.198	0.542	0.961	0.498	0.662	0.112	0.039	0.039	0.773	0.811

Results were expressed as Spearman's correlation coefficient r and statistical significance p. T2DM, type 2 diabets mellitus; BMI, body mass index; FBS, fasting blood glucose; TG, triglyceride; HDL, high density lipoprotein. LDL, low density lipoprotein.

DISCUSSION

Lower plasma levels of adiponectin relative to the normal controls were documented in human subjects with obesity, insulin resistance and type 2 diabetes in several cross sectional studies [12, 13]. Results from a few prospective studies suggest that low adiponectin level is predictive of insulin resistance or diabetes [14, 15]. Although the association of low adiponectin concentration and type 2 diabetes is rather well studied, only few studies investigated adiponectin level in pre-diabetes (e.g. impaired glucose tolerance and impaired fasting glucose [16].

In Pearson's correlation analysis a positive correlation was observed between adiponectin and age in the control subjects. A study by Spiegelman BM.,

(2001) observed that adiponectin was positively associated with age [17].

In our study a negative correlation was observed between adiponectin and BMI in IGT subjects. Recently in a study on lean and obese Saudi females with T2DM subjects found that adiponectin was negatively correlated with BMI [18]. Previous studies in Japanese individuals have shown that the adiponectin concentration was negatively correlated with body mass index BMI; accordingly, it was lower in obese subjects than in lean subjects. In our study no significant correlation was observed with adiponectin and WHR in any of the study groups instead of the negative association with BMI in IGT subjects.

In the present study post prandial glucose had a positive correlation with adiponectin in control subjects. Our study demonstrated a positive correlation with adiponectin and serum HDL and a negative correlation with serum LDL in T2DM subjects. This finding are consistent with the study [19]. They suggested that high adiponectin concentration and its anti-atherogenic function are associated with increased HDL levels in blood.

A significant association was found between adiponectin and β-cell function (HOMA%B) and not insulin resistance [20]. In a study on GDM subjects, they also found a positive association between β-cell dysfunction and serum adiponectin. They observed that hypoadiponectinaemia was associated with both insulin resistance and beta cell dysfunction in GDM and adiponectin and was considered as an important factor potentially linking insulin resistance and beta cell dysfunction in the pathogenesis of type 2 diabetes.

In our study no male female difference was observed regarding the adiponectin value among the study subjects. This result contradicts with some other studies, they observed the association between adiponectin and type 2 diabetes was stronger in women than in men [21]. It is remarkable that, despite their higher body fat percentage, women appear to have higher adiponectin levels than men. Previously, this difference could not be explained by differences in fat distribution [22]. Realizing established and possible role of adiponectin in pathobiochemistry of obesity-related disorders, it was suggested that obese males are at higher risk of hypoadiponectinemia with subsequent metabolic disturbances, than females are. Decreased serum adiponectin concentration, as independent metabolic risk factor may identify susceptibility for pre-diabetes in males specially.

CONCLUSIONS

In conclusion we can say that BMI has a negative association with adiponectin in IGT subjects. erum LDL has a negative association and HDL has a positive association with adiponectin in IGT and T2DM subjects. Hyperglycemia has a positive association with hypoadiponectinaemia in the study subjects.

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