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Original Article

Evaluation of blood glucose levels and glycated hemoglobin in newly diagnosed type 2 Diabetes Mellitus without complications

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ABSTRACT

Introduction: Diabetes mellitus is not one disease, but rather is a heterogeneous group of multifactorial, polygenic syndromes characterized by an elevated fasting blood glucose (FBG) caused by a relative or absolute deficiency in insulin. Objective: The main objective of this study was to assess the relationship between blood glucose levels and HbA1c in control and newly diagnosed diabetic patients without complications. Methods. The present study was conducted on 75 newly diagnosed type 2 Diabetes mellitus patients (female- 25, male- 50) and the control group consisted of 60 healthy individuals (female-24, male-36). Blood samples were collected in plain vacutainers without any anticoagulant and whole blood collected with EDTA from diabetic patients and control subjects for the estimation of various biochemical parameters. Blood glucose estimation was done by GOD-POD method and HbA1c, creatinine, urea, total protein and albumin were estimated by kit method (ERBA diagnostics Mannheim GmbH). Statistical analysis was done by unpaired t-test and Pearson correlation coefficient. Results: FBS (148.60 ± 7.8), PPBS (193.92 ± 12.30) mg/dl and HbA1c (6.51 ± 0.85) % was significantly higher in type 2 diabetes patients ($p<0.0001$) as compared to control subjects FBS (95.23 ± 6.3), PPBS (121.35 ± 9.50) mg/dl and HbA1c (4.58 ± 0.32) % ($p<0.0001$). FBS was found to be highly correlated ($r=0.774$, $p<0.001$) with HbA1c as compared to PPBS ($r=0.427$, $p<0.05$) in newly diagnosed diabetic patients. Conclusions: Our study showed that FBS is highly correlated to HbA1c as compared to PPBS.

Introduction

Diabetes mellitus is not one disease, but rather is a heterogeneous group of multifactorial, polygenic syndromes characterized by an elevated fasting blood glucose (FBG) caused by a relative or absolute deficiency in insulin [1]. Excess circulating glucose in diabetes is a reactant molecule, involved in the glycosylation of various biomolecules like haemoglobin [2]. Glycated haemoglobin (HbA1c) is formed in a non- enzymatic glycation pathway by hemoglobin's exposure to plasma glucose and binding to the N-terminal of valine of β chain of haemoglobin [3]. Level of glycated haemoglobin (Hb) depends upon lifespan of Red blood cell (average 120 days) and the blood glucose concentration. Glycated haemoglobin (HbA1c) expressed as a % of total blood haemoglobin concentration gives a good retrospective assessment of the mean plasma glucose concentration during the preceding 6-8 weeks [2,4]. Studies on chronic complications of diabetes established the role of glycated haemoglobin (HbA1c) as a marker of evaluation of long term glycemic control and risk for chronic complications [5]. Since fluctuations of fasting plasma glucose (FPG) and postprandial plasma glucose (PPPG) could affect HbA1c the present study was designed to assess the relationship between the blood glucose levels and HbA1c in newly diagnosed diabetic patients without complications.

Materials & Methods

The criteria used for selection of both diabetes and normal controls were performed by well-established diagnostic criteria as recommended by WHO. The present study was conducted on 75 type 2 Diabetes mellitus patients without complications (female- 25, male- 50) with mean age of 38.8 ± 3.4 years and the control group consisted of 60 healthy individuals (female-24, male-36) with mean age of 36.2 ± 2.8 years. Diabetic subjects were only on oral hypoglycemic drugs. The study groups (patients and control) were non-smokers and non-alcoholics and were not suffering from any other chronic disease. The study was approved by the Institute Ethics Committee, Index Medical College Hospital and Research Centre, Indore, India and informed consent was obtained from all the cases and control subjects. Blood samples were collected in plain vacutainers without any anticoagulant and whole blood collected with EDTA from diabetic patients and control subjects for the estimation of various biochemical parameters.

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1. Fasting and postprandial blood sugar was estimated by method of GOD-POD Trinder P. [6]

2. HbA1c by kit method (ERBA diagnostics Mannheim GmbH).

3. Creatinine by kit method (ERBA diagnostics Mannheim GmbH).

4. Urea by kit method (ERBA diagnostics Mannheim GmbH).

5. Total Protein by kit method (ERBA diagnostics Mannheim GmbH).

6. Albumin by kit method (ERBA diagnostics Mannheim GmbH)

All reagents and chemicals used were of analytical grade and were purchased from Merck Chemical Co., Germany. All data were expressed as mean ±SD. Unpaired student's t-test was used for between group comparisons. Differences were considered of statistical significance when the p-value was $p<0.05$. Pearson correlation coefficient (r) was calculated for the comparison of blood sugars (fasting and post prandial) with respect to HbA1c.

Result

The study was conducted on 75 newly diagnosed type 2 diabetes mellitus patients without complications (female- 25,male-50) mean age 38.8 ± 3.4 years and the control groups consisted of 60 individuals (female-24,male-36) mean age 36.2 ± 2.8 years. The demographic and biochemical parameters in type 2 diabetes mellitus patients and control subjects are depicted in Table 1. FBS (148.60 ± 7.8), PPBS (193.92 ± 12.30) mg/dl and HbA1c (6.51 ± 0.85) % was significantly higher in type 2 diabetes patients ($p<0.0001$) as compared to control subjects FBS (95.23 ± 6.3), PPBS (121.35 ± 9.50) mg/dl and HbA1c (4.58 ± 0.32) % ($p<0.0001$) as shown in Table 1.

Pearson correlation coefficient for FBS and PPBS with respect to HbA1c has been depicted in Table-2. FBS was found to be highly correlated ($r=0.774$, $p<0.001$) with HbA1c as compared to PPBS ($r=0.427$, $p<0.05$) in newly diagnosed diabetic patients.

Table 1. Demographic and Biochemical Parameters in Type 2 Diabetes Mellitus Patients & Control Subjects

Parameters	Type 2 Diabetes Mellitus Patients	Controls	P-value
	N=75	N= 60	
Age (years)	38.8 ± 3.4	36.2 ± 2.8	-
Sex (M/F)	50/25	36/24	-
BMI (kg/m^2)	28.5 ± 2.50	24.6 ± 1.80	<0.001
Duration of disease (years)	5.20 ± 1.26	-	-
Hemoglobin (gm/dl)	12.78±1.32	13.21±1.51	NS
FBS (mg/dl)	148.60 ±7.8	95.23 ± 6.3	<0.0001
PPBS (mg/dl)	193.92 ± 12.30	121.35 ± 9.50	<0.0001
HbA1c (%)	6.51 ± 0.85	4.58 ± 0.32	<0.0001
Serum Creatinine (mg/dl)	1.05±0.26	0.95±0.32	<0.05
Urea (mg/dl)	31.6±5.8	28.1±6.4	<0.001
Total Protein (gm/dl)	6.4± 1.0	7.2±1.2	<0.0023
Albumin (gm/dl)	3.75±0.48	4.53±0.42	<0.0032

Values are expressed as mean ± S.D N= number of subjects; 'P' <0.05 was considered significant. NS= not significant

Table 2: Pearson correlation coefficient for fasting blood sugar and post prandial blood sugar with respect to HbA1c

	HbA1c	
Parameter	Pearson correlation coefficient (r)	P-value
Fasting blood sugar	0.774	<0.001
Post prandial blood sugar	0.427	<0.05

Values are expressed as mean ± S.D N= number of subjects; 'P' <0.05 was considered significant. NS= not significant

Discussion

Glycemic control is an important aspect in managing diabetes in order to prevent acute or chronic complications of diabetes mellitus. The HbA1c assay has become the most commonly used measure of chronic glycemia in epidemiological studies, clinical trials and the management of diabetes [7]. It is the basis of treatment guidelines and is used universally to adjust therapy [8, 9] and to reduce the risks of microvascular complications. Our results indicate highly significant correlation in FBS and HbA1c levels in newly diagnosed diabetic patients as compared to normal subjects. These results are in accordance with the study of Bonora et.al. 2001 [10]. Similar conclusions have also been drawn by Peter et.al. 2006 [11] and Goudswaard et.al. 2004 [12]. On the contrary the study by Rosediani et.al. 2006 [13] has stated that HbA1c correlated more closely to post prandial blood sugar than to preprandial blood sugar.

Conclusion

Thus from this study we conclude that HbA1c is more closely related to FBS as compared to PPBS. Therefore measures should be taken to reduce both blood glucose levels but more focus should be on fasting hyperglycemia so as to prevent acute and chronic complications.

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