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Original Article

STUDY ON GLYCEMIC STATUS IN SIBLINGS OF DIABETIC PARENTS

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ABSTRACT

Introduction: As per Indian council of medical research survey, incidence of diabetes in 1975 was around 2.1% which increased to 4.5% by 1990 and with the present trend is likely to reach around 6.8% in this millennium. Offspring of patients with Non insulin depended diabetes mellitus have a 40-60% chance of developing type 2 diabetes and increased frequency of impaired glucose tolerance. The siblings of diabetic parents are at high risk than siblings of non diabetic parents. The glycemic status plays a crucial role and it is a good indicator to alert the offspring's of diabetic parents. **Materials & Methods:** The study was carried in department of Biochemistry on 30 control cases (23 Males 07 Females) of siblings of non diabetic parents (Group I) and 60 cases (42 Males, 18 Females) of siblings of diabetic parents (Group II) of same aged group. The glycemic status of both groups is studied by estimating the Glucose tolerance test and glycated haemoglobin. **Results:** Siblings of diabetic parents were further divided into two groups, normal glucose tolerance group and impaired glucose tolerance group. The Body mass index, fasting blood glucose, peak blood glucose and glycated haemoglobin levels were shown significant difference in siblings of diabetic parents with impaired glucose tolerance.

Keywords: *Blood glucose, Siblings, Diabetic parents, Glycemic status*

INTRODUCTION

Diabetes mellitus is third leading cause of death after heart disease and cancer in many developed countries [1]. Forward looking, it is a modern threat to the public health and most prevalent non communicable disease in the world [1, 2].

The greatest population are being suffering with diabetes globally, based on the survey done by WHO in 1995, around 135 million adults are diabetic and it is anticipated to advance to 300 million in 2025 [3]. As per the statistical survey of the Indian Council of Medical Research-India Diabetes (ICMR-INDIAB) there are 62.4 million living with diabetes in India, incidence of diabetes in 1975 was close to 2.1% which changed magnitude to 4.5% by 1990 and with the exhibit trend is probably to reach around 6.8% in this millennium [4]. Out of 62.4 million, 2% diabetics are insulin dependent and 98% are non insulin dependent [1, 5].

Up on the known diabetic population there are several individuals who are unaware of the diabetic which add up rigorousness to the incidences of diabetes. Growing urbanization and changing life style lead to physical inactivity thus leading to overweight and obesity, which leads to insulin resistance [6]. This has resulted in an unprecedented rise of diabetes to epidemic proportion during last few decades in our country [4, 6].

As we aware of that 98% of diabetic cases are of NIDDM. So, suitable model for the study of natural history of NIDDM is the offspring of diabetic parents [7]. Offspring of patients with NIDDM have a 40-60% chance of developing type 2 diabetes and increased frequency of impaired glucose tolerance [8].

The plasma sugar levels in OGTT in normal persons and diabetic patients (based on WHO recommendations 1999).

Time	Normal	Criteria for Diagnosing diabetes	Criteria for Diagnosing IGT
Fasting	<110 mg/dl	> 128 mg/dl	< 110 mg/dl
1hr. (peak) After glucose	< 160 mg/dl	Not prescribed	Not prescribed
2 hr. after glucose	< 140 mg/dl	> 200 mg/dl	< 140 mg/dl

This study was undertaken to know the glycemic status in siblings of diabetic parents. Glycemic status can be better studied by glucose tolerance test and glycated Haemoglobin. Glucose tolerance can be reported normal or impaired depending upon their genetic inheritability found in siblings of diabetic parents. Delay in the development of diabetes and its complications can be achieved by eliminating environmental risk factors and taking early precautions. Thus by studying siblings of diabetic parents can become a good indicator of onset of NIDDM.

MATERIALS AND METHODS

The current study was carried out in department of Bio-Chemistry, Government Medical College, Azamgarh. The diabetic patients were identified from the registry of diabetic clinic and accompanying siblings were taken as study cases. Total 60 cases of siblings of diabetic parents are counted for the study and 30 control cases were taken from siblings of non diabetic parents of same aged group.

The study subjects are divided into groups. Group I was siblings of non diabetic parents (control). Group II was siblings of diabetic parents (case), further it is divided into II A & II B, II A was siblings of diabetic parents with normal glucose tolerance and II B was siblings of diabetic parents with impaired glucose tolerance. Glycemic status of siblings of diabetic parents and siblings of non diabetic parents is performed by Oral Glucose Tolerance Test (by estimation of blood glucose) and estimation of Glycated haemoglobin. The Oral Glucose Tolerance Test (OGTT) is performed by using the Hexokinase method. The estimation of glycated haemoglobin was done by NYCOCARD.

RESULTS

The two Biochemical parameters were taken for consideration to know the glycemic status of the siblings which includes oral glucose tolerance test and glycated haemoglobin.

As a whole 60 siblings of diabetic parents were studied and 30 siblings of non diabetic and healthy parents were studied and matched as a control.

Table 1: Showing division of groups, Sex, Age and Body Mass Index (BMI) of siblings of both case and control

Group		**No. Of Subjects**	**Sex**		**Age**	**BMI**			
						Mean	**SD**	**P**	
I		30	M	23	25.4	22.2	2.9	0.44	---
			F	07	25.2				
II	A	36	M	23	26.5	20.95	2.7		0.001
			F	13	25.3				
	B	24	M	19	29.6	25.7	3.3		
			F	05	28.5				
			P<0.05 considered as Significance; M-Male; F- Female						

*BMI – Body Mass Index

*SD – Standard Deviation

The mean age in group I i.e. siblings of non-diabetic parents is 22.2 and 25.2 respectively in males and females, not much deviation is observed between mean age of group II A & II B (Table – 1). The mean difference of Body Mass Index (BMI) in group I and II is statistically not significant ($p \geq 0.05$), Group II B shows increase in Body Mass Index as compared to group II A and it is statistically significant ($p<0.05$). After analysis it was observed that the mean values of Fasting blood glucose (FBG), Peak blood glucose (PBG), Glycated Haemoglobin (GH) was significantly more in group II compare to group I ($P \leq 0.05$) (Table – 2). The mean differences between Group II A and II B was also shown the significant differences in all the parameters (Table – 3).

Table 2: Showing the statistical difference of glucose tolerance and glycated haemoglobin in group I and II

Parameter	Study subject	Mean mg (%)	SD	t	P
FBG	I	83.7	12.0	2.4	0.05
	II	91.0	16.3		
PBG	I	122.5	19.6	3.8	0.05
	II	142.4	29.3		
GH	I	4.86	0.94	2.39	0.05
	II	5.49	1.59		
		P≤0.05 considered as Significance			

*FBG – Fasting Blood Glucose

*PBG – Peak Blood Glucose

*GH – Glycated Haemoglobin

*SD – Standard Deviation

Table 3: Showing mean, SD, t value and P value of group II A and II B for FBG (Fasting Blood Glucose), PBG (Peak Blood Glucose), GH (Glycated Haemoglobin)

Parameter	Study subject	Mean mg (%)	SD	t	P
FBG	II A	80	10.5	12.8	0.001
	II B	107.6	5.9		
PBG	II A	124	21.5	10.4	0.005
	II B	170.2	12.6		
GH	II A	4.31	0.69	17.7	0.005
	II B	7.27	0.62		
		P≤0.05 considered as Significance			

*FBG – Fasting Blood Glucose

*PBG – Peak Blood Glucose

*GH – Glycated Haemoglobin

*SD – Standard Deviation

DISCUSSION

The observations that we found in current study to know the glycemic status of siblings of diabetic parents by knowing the Body mass index, fasting and peak blood glucose, glycated haemoglobin. All the parameters were compared statistically for its significance with control and observed significant nature of changes in different parameters.

Glucose tolerance test is a good indicator for assessing potential diabetics by measuring their blood glucose levels. Changes from normal to impaired glucose tolerance had bearings on number of complications and future progression into diabetes. Glycosylated Haemoglobin is a normal haemoglobin component and monitors diabetic patient's complicance [9]. Glycosylated haemoglobin concentration reflects the glycemic control over previous 1 to 2 months [4].

In the present study the mean fasting blood glucose level in group I and group II were 83.73 mg % and 91.05 mg % respectively and they were statistically significant (Table - II). The mean peak blood glucose level in group I and II were 122.5 mg% and 142.4 mg% respectively. The mean differences in group II A, group II B of fasting and peak blood glucose levels are statistically significant. The glycated haemoglobin showed significant differences in between siblings of non-diabetic parents (group I) and siblings diabetic parents (group II), as well as in siblings of diabetic parents with normal glucose tolerance (group II A) and siblings of diabetic parents with impaired glucose tolerance(II B).

Ganda OP et al [10] (1985) studied 150 offspring's of type 2 diabetic parents and 67 normal control subjects for their plasma lipids and glucose tolerance. They found that the fasting blood glucose and insulin responses were significantly elevated in the offspring's of diabetic group compared with controls and offspring's of non diabetic group. The study of Leslie et al [11] (1986) reported that offspring's of Non Insulin Dependent Diabetic Mellitus (NIDDM) had significantly higher fasting glucose concentrations and Glycosylated Haemoglobin (HbA_{1c}). Gulli et al [12] (1992) found that fasting plasma glucose concentration was similar in siblings of non-diabetic parents and siblings of NIDDM. After glucose ingestion both control and cases had normal oral glucose tolerance. Kuo-Liong Chien [13] found abnormal glucose tolerance in all 8 offspring's of diabetic patients over the age of 50 years and interpreted this as evidence that all offspring's of diabetic patients will eventually develop diabetes.

This can be attributed to genetic predisposition, increased body mass index (25.75%) in our study. This genetic predisposition in our study can be correlated with maternally inherited mitochondrial point mutations [6]. Hyper insulinemia and hyper glucagonemia may be attributed to glucose in tolerance in our case but our study did not estimated insulin and glucagons levels confirmation remains in conclusive [14]. We may state that offspring of diabetic parents were associated with moderate disorders typical of NIDDM such as insulin resistance, hyper insulinemia, dyslipidemia and high body fat content prior to the onset of glucose intolerance.

CONCLUSION

The findings of present study support the view that siblings of diabetic parents were good source samples to study the course of NIDDM and its familial inheritance. In the present study the siblings of diabetic parents who have impaired glucose tolerance can invite the development of diabetics in future and its complications due to deranged lipid profile if they be taken as 'warning signal' for these subjects. Lastly, we conclude that high-risk siblings of diabetic parents can delay diabetes and its complications by controlling blood sugar, diet, regular exercise and changing life style, and it was the major concern of this work.

Conflict of interest: Nil

REFERENCES

1. Mc Keigue PM, Shah B and Marmot MG. Relationship of central obesity with insulin resistance with high diabetic prevalence and cardiovascular risk in south Asians. Lancet 1991:337:382-86.
2. Vasudevan D.M & sreekumari S, Text book of biochemistry, Edition 3 pg no: 134-36.
3. Dahdwal D., Ahluwalia S.K., Das Gupta D.J., prevalence of NIDDM in the general population (>40 yrs) in shimla. Indian J. Med. Sci. 1997:57:459-64.
4. Koenig RJ, Paterson CM, Jones RL, Saddek C et al Correlation of glucose regulation and HbA1C in diabetes. N Eng J Mzd 1976:295:417.
5. Cooke A.M., Fitzgerald M.G. Malins J. and pyke A.A.:Diabetes in children of diabetic couples Br.Med J.2:674, 1996.
6. Jackson W.P.O: A clinician looks at genetics problems in diabetes proc. 7th Congress of the IDF Bundes Aires, R R. Rodniquner and J. Vallance ower in the congress series No.971;231
7. Tattersall RB, Fajans SS, Arbor A, Prevalence of Diabetes and glucose intolerance in 199 offspring's of thirty-seven conjugal diabetic parents. Diabetes 1975; May 24 (5); 452-62.
8. Foss C.N., Vestbo E., Froland A., Gjessing H.J., Mogensen C.E. and Damsgaard E.M. Normal blood pressure and preserved diurnal variation in offspring of type 2 diabetic patients characterized by features of the metabolic syndromes: The fredericia study. Diabetes care 2000 Mar.23 (3):283-89.
9. Albrink M.J and Davidson P.C.: Impaired glucose tolerance with hypertriglyceridemia. J Lab clin Med1996; 67: 573-84
10. Ganda OP, Soeldner S and Gleason RE. Alternations in Plasma lipids in the presence of Mild Glucose intolerance in the offspring to two type II diabetic parents, Diabetes care 1985; 8 (3):254-60.
11. Leslie RDG, Volkmann HP, Poncher M, hanning I, Orskov H, Alberti KGMM. Metabolic abnormalities in children of non-insulin dependent diabetes, British Medical Journal 1986; Oct 293:840-42.
12. Gulli G, Ferranninini E, Michael S, Haffner S and DeFronzo RA. The metabolic profile of NDDM is fully established in glucose-tolerant offspring of two Mexican-American NIDDM parents. Diabetes 1992; decem 41: 1575-86.
13. Kuo-Liong Chien A,C, Hsiu-Ching HSuC, Ta-Chensu; Wei-Tien Changd, Pei-Chun chenb, Ming Fong chenc and Yuan-The Leec. Siblings and parental history in type 2 diabetes risk among ethnic Chinese: the chin-Shan community cardiovascular cohort study. Evr J cardiovasc Prev Rehabil 2008; 15:657-62.
14. Bierman EL and Porte DJr: Corbohydrate intolerance and lipemia. Ann Intern Med1968; 68: 926-33