ORIGINAL RESEARCH ARTICLE

The study of Insulin Resistance in the Off Springs of Diabetics and Non Diabetic Patients

Ganesh Manoorkar[1], Suvarna Tale [2]

Associate Professor, Department of Biochemistry, Grant Government Medical College, Mumbai[1], PG student Department of Biochemistry, Grant Government Medical College, Mumbai-400008, Maharashtra, India[2]

Abstract:

Introduction:

Insulin resistance is one of the main cause in the pathogenesis of the development of type- 2 diabetes mellitus. Elevated insulin levels and insulin resistance may be present several years prior to the development of hyperglycaemia. Hence the diagnosis of insulin resistance at the initial stages in risk group people could be used as an effective measure to prevent type 2 diabetes mellitus and its outcome, including reduction in morbidity and mortality. Though type 2 diabetes mellitus has multifactorial aetiology, genetic factor plays an important role in the development of diabetes mellitus. So we have tried to establish relation between genetic factor and insulin resistance by studying the insulin resistance in off springs of diabetics and non diabetics patients.

Aims and objectives:

Estimation of insulin levels in the off springs (non diabetics) of diabetics and non diabetics patients.

Comparision of insulin resistance in the off springs (non diabetics) of diabetics and non diabetics.

To find the relation between insulin resistance and genetic factor.

Material and method:

This study was carried out in the department of Biochemistry Grant Government Medical College Mumbai. Total 100 non diabetic people were included in the study of age above 30 years. These are divided into two groups as-

Group-I includes 50 off springs (I^{st} degree relatives) of non diabetic people.

Group-II includes 50 off springs (I^{st} degree relatives) of diabetic people.

The fasting plasma glucose and serum insulin levels are estimated in the above two groups. The insulin resistance was calculated by using HOMA-IR model.

Result:

Fasting plasma glucose, serum insulin level and insulin resistance is significantly increased in group-II people as compared to group-I people.

Conclusion:

There is a strong relation between genetic factor and insulin resistance which exist prior to the development of diabetes mellitus. The people of group-II are susceptible for the development of diabetes mellitus. If these people are identified and managed early we may prevent or may delay the development of the type 2 diabetes mellitus in these people.

Key words:

Type 2 diabetes mellitus, Insulin resistance

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Address for correspondence:

Dr. Ganesh. S. Manoorkar, Associate Professor, Department of Biochemistry, Grant Government Medical College, Mumbai -400008. Maharashtra, India.

Email: drganeshmanoorkar@yahoo.in, Mobile No: 9823172

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Introduction:

Diabetes mellitus is a metabolic disorder as a result of either insulin resistance and/or insulin deficiency/ or both. It is a complex disease where the carbohydrate and fat metabolisms are affected predominantly. Type 2 diabetes mellitus is considered as one of the most predominant type of diabetes mellitus since it represent majority of cases. Though type 2 diabetes mellitus has multifactorial aetiology, genetic factor plays an important role in the development of type 2 diabetes mellitus[1]. In such people elevated insulin levels and insulin resistance may be present several years prior to hyperglycemia.

Insulin resistance:

Insulin resistance is defined as the diminished ability of cells to respond to the action of insulin in transporting glucose (sugar) from the bloodstream into muscle and other tissues.

If we eat or drink any food containing glucose (or the digestible carbohydrates) plasma glucose level increases. In normal persons, the elevated blood glucose level stimulates beta (β) cells in the Islet of Langerhans, of pancreas, to release insulin into the blood. The insulin, in turn, makes tissues in the body (primarily skeletal muscle cells, adipose tissue) to absorb glucose, and thereby lower the blood glucose level. When blood glucose level decreases insulin output also decreases. In an insulin-resistant person, normal levels of insulin do not have the same effect in controlling blood glucose levels. During the compensated phase of insulin resistance insulin levels are higher, and blood glucose levels are still maintained.

Thus diagnosis of insulin resistance at the initial stages in these risk group people may be used as an effective measure to prevent type 2 diabetes mellitus and its outcome, including reduction in morbidity and mortality. So we have tried to establish relation between genetic factor and insulin resistance by studying the insulin resistance in off springs of diabetics and non diabetic patients.

Aims and Objectives:

1) Estimation of plasma glucose and insulin level in the off springs (non diabetics) of diabetics and non diabetics patients.
2) Comparison of insulin resistance in the off springs (non diabetics) of diabetics and non diabetics.
3) To find the relation between insulin resistance and genetic factor.

Material and Methods:

This study was carried out in the department of Biochemistry Grant Government Medical College Mumbai.

Total 100 non diabetic people were included in the study of age above 30 years. These are divided into two groups as-

Group-I include 50 off springs (I^{st} degree relatives) of non diabetic people.

Group-II includes 50 off springs (Ist degree relatives) of diabetic people.

The fasting plasma glucose and serum insulin levels are estimated in the above two groups. The insulin resistance was calculated by using HOMA-IR model.

Ratio of males and females kept same in all the groups.

Informed consent will be obtained from all the study participants and the ethical

committee of our tertiary care hospital approved the study.

Exclusion criteria:

The people with known diabetes mellitus, presence of risk factors of diabetes mellitus, obesity, hypertension, liver disease, kidney disease, thyroid disease, etc.

Statistical analysis:

The statistical analysis will be done using SPSS for Windows, version 17. Student's t-test and Pearson correlation test will be used for the analysis of p value and correlation of scale variables. Results with $p < 0.05$ will be accepted as statistically significant. Data will be expressed as mean ± SD.

Estimation of Fasting Plasma Glucose (mg/dl):

Plasma from the fluoride bulb was used for the estimation of plasma glucose.

Method:

GOD-POD method (Glucose oxidase and peroxidase method):

The WHO diagnostic criteria for Diabetes mellitus:

Fasting plasma glucose ≥ 126mg/dl or

Post meal plasma glucose ≥ 200 mg/dl

Estimation of Serum Insulin (uIU/ml):

Serum from the plain bulb was used for the estimation of serum Insulin.

Method:

Serum insulin concentrations were measured by means of a Chemiluminescence Immunological Assay.

Diagnostic criteria for increase in serum Insulin:

The proposed reference interval for normal fasting insulin levels was 6 to 27 uIU/ml[2].

Assessing Insulin Resistance: HOMA-IR (Homeostasis model assessment of insulin resistance):

HOMA-IR was developed by Matthews[3] as a method for estimating insulin sensitivity from fasting serum insulin and fasting plasma glucose.

Insulin sensitivity was calculated by the homeostasis model assessment method (HOMA-IR), which was calculated by following formula:

HOMA-IR = Fasting Plasma Glucose (mg/dL) × fasting Insulin (uIU/ml) / 405.

Diagnostic criteria for Insulin resistance:

Subjects with a HOMA-IR (homeostasis model assessment of insulin resistance) level above the 3.5 of the present study population were defined as having insulin resistance [(4)].

Low HOMA values indicated high insulin sensitivity, and high HOMA values indicated low insulin sensitivity.

Results:

Table – 1 Table showing the comparison of all parameters in Group-I and Group-II in study population:

Parameters	Group-I	Group II	P value
Mean Plasma fasting glucose level (mg/dl)	73.67 ± 10.23	89.16 ± 17.68	P < 0.05
Mean Serum Insulin level (uIU/ml)	14.41 + 4.18	28.80 ± 11.23	P < 0.05
Mean value of HOMA-IR	2.57	6.34	P < 0.05

Group-II shows significantly higher mean plasma glucose, higher mean serum insulin level and higher insulin resistance.

Discussion:

We have found that mean fasting plasma glucose is higher in group-II (89.16 ± 17.68 mg/dl) as compared to group-I (73.67 ± 10.23 mg/dl).

Renuka Pangaluri et al found higher mean fasting plasma glucose (109.2 ± 14.3 mg/dl) in patients as compared to mean fasting plasma glucose (85.4 ± 12.4 mg/dl) in normal controls[5]. They suggest glucose uptake in the muscles and adipose tissue is affected due to insulin resistance.

Eman M et al also found higher mean fasting plasma glucose as 5.7±0.8 5 (mmol/l) in insulin resistance people and 5.4 ± 0.7 (mmol/L) in non insulin resistance people[6].

We have found mean serum insulin is higher in the group-II (28.80 ± 11.23 uIU/ml) as compared to group-I (14.41 + 4.18 uIU/ml).

Recently, a genomewide association study (GWAS) has found that the mutation in the KCNQ1 gene related to insulin secretion abnormality. It is an important disease-susceptible gene associated with the pathogenesis of diabetes in Asian ethnic groups including the Japanese [1].

Eman M et al found higher mean insulin level which is 11.2 (8.3 - 14.2) (mU/l) in insulin resistance people than non resistance people in which mean insulin level is 6.1 (4.2 - 8.4) (mU/l)[6].

Richard Mack Blanche et al found lower mean fasting insulin level in control as 15.8μIU/mL and higher in type 2 diabetes as 29.1 μIU/mL[2].

Renuka Pangaluri et al found higher mean fasting plasma insulin level as 25.72 ± 5.29 (μIU/mL) in patients and 12.45 ± 2.2 (μIU/mL) in controls[5].

We have also found mean HOMA-IR is higher in group-II than group-I.

Reizo Baba etal found insulin resistance in non obese adolescents i.e. below 40 years [7].

Conclusion:

There is a strong relation between genetic factor and insulin resistance which exist prior to the development of diabetes mellitus. The people of group-II are susceptible for the development of diabetes mellitus. If these people are identified and managed early we may prevent or may delay the development of the type 2 diabetes mellitus in these people.

Conflict of interest: None to declare

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