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RELATIONSHIP OF FASTING BLOOD GLUCOSE, HEMOGLOBIN A1C (HBA1C) AND HOMEOSTASIS MODEL ASSESSMENT OF INSULIN RESISTANCE (HOMA IR) IN DIAGNOSED TYPE 2 DIABETES PATIENTS

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ABSTRACT

Background: Diabetes is a group of metabolic diseases characterized by hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. This study is an attempt to determine the correlation between HbA1c and HOMA IR in type 2 Diabetes patients.

Material And Methods: The study had 240 participants between the age groups of 20-65 years included 80 males and 40 females in diabetic group whereas the control group included 78 males and 42 females. HbA1c was measured by turbidimetric Method. HOMA-IR was calculated using the following formula: HOMA-IR = fasting serum glucose (mg/dL) × fasting serum insulin value (μU/mL)/405.

Results: Data was represented as mean ± standard deviation. There was a significant positive correlation between HbA1c, Fasting Glucose, and HOMA IR. Patients with HbA1c value greater than 7.0% had significantly higher value of HOMA IR and compared with patients with an HbA1c value up to 7.0%.

KEYWORDS : Diabetes Mellitus, Glycated hemoglobin (HbA1c), HOMA IR, Cardiovascular disease (CVD).

INTRODUCTION

Diabetes mellitus (DM) is a common chronic non -infectious disease, which can make the body continuously hyperglycemia and long-term metabolic disorder, and lead to the damage, dysfunction and failure of the whole-body tissues and organs, especially the eyes, kidneys, Cardiovascular system and nervous system[1].Type 2 diabetes (T2DM) accounts for >90% of cases and is characterized by insulin resistance, often accompanied by relative insulin deficiency[2]. The main pathophysiological defects responsible for type 2 diabetes mellitus (T2DM) include cell dysfunction and decreased insulin sensitivity. In case of Insulin resistance, there is progressive loss of beta – cell function and there are different factors including hyperglycemia and elevated fatty acid which accelerate the deterioration of beta cell[3].

Insulin resistance is the earliest metabolic abnormality detected in subjects destined to develop type 2 diabetes. In response to insulin resistance, the b-cell appropriately increases insulin secretion and normal glucose tolerance (NGT) is maintained. However, when b-cell failure ensues, glucose intolerance develops. Initially, this is manifest as impaired glucose tolerance (IGT) and subsequently as overt diabetes. Thus, impaired b-cell function is an essential condition in the development of type 2 diabetes[4].

In blood, Analysis of glycated hemoglobin (HbA1c) provides evidence about an individual's average blood glucose levels during the previous two to three months, which is the predicted half-life of red blood cells and the HbA1c is now recommended as a standard of care (SOC) for testing with monitoring diabetes, specifically in the type 2 diabetes[5].

In 1997, the American Diabetes Association (ADA) revised its criteria for the diagnosis of type 2 diabetes and determined that subjects with fasting plasma glucose (FPG) >126 mg/dL and 2-h plasma glucose ≥ 200 mg/dL are considered to have type 2 diabetes. These cut points were chosen on the basis of the increased incidence of diabetic retinopathy rather than on the presence of metabolic abnormalities (i.e., insulin resistance and b-cell dysfunction) that are responsible for type 2 diabetes[6].

Yunxia Lu in their study found New-onset type 2 diabetes and, particularly, diabetes with rising HbA1c seem to be independent risk factors for pancreatic cancer. The relation between different anti-diabetic medications and pancreatic cancer seems to vary in strength, with the highest risk among users of insulin[7].

T2DM is currently one of the most common chronic diseases, affecting approximately 415 million adults in the world and its incidence has risen dramatically in recent years, especially in middle-aged and elderly. The progressive deterioration of islet β-cell function and insulin resistance are the main pathophysiological factors of adult type 2 diabetes mellitus[8].

MATERIALS AND METHODS

The present study is prospective study, will be carried out in the Department of Biochemistry and Department of General Medicine, Index Medical College, Hospital & Research Center (IMCHRC), Indore. The cases were selected from those who attended the medicine outpatient department (OPD) during the period of 1 year (January 2019 to December 2019) at IMCHRC, Indore.

INCLUSION CRITERIA & EXCLUSION CRITERIA:

All cases and control were included with age group of 18-65 years in both male and female. Cases and healthy control suffering from immune deficiency disease were included as patients with known case of hypothyroidism, Cushion's syndrome was excluded from the study. Patients diagnosed with the Type 2 diabetes were included whereas Type I diabetes patients were excluded from the study. In the selection of patients, patients below age of 18 years or known or suspected with pregnancy were excluded.

Sample Collection

After 12 hours overnight fast, 6.0 ml of blood was collected from each subject by venipuncture with standard blood collection technique in a plane vial for serum separation, sodium fluoride vial for plasma and EDTA vial for HbA1c estimation. Plasma was collected again after two hours of post meal for the postprandial glucose estimation.

Parameters Measured

The following parameters were measured in this study:

1. Serum fasting blood sugar (FBS)
2. HbA1c.
3. Insulin
4. HOMA IR

Serum FBS was measured by GOD-POD method with the help of Randox RX, a fully automatic analyzer. HbA1c was measured by turbidimetric immunoassay with the help of HPLC Method. Serum insulin was measured by ELISA method followed by instruction provided by manufacturer. Our study used the homeostasis model assessment of insulin resistance (HOMA-IR) as the diagnostic criteria for insulin resistance and HOMAIR ≥ 2.5 was identified as an indicator of insulin resistance. HOMA-IR was calculated using the following formula: HOMA-IR = fasting serum glucose (mg/dL) × fasting serum insulin value (μU/mL)/405[9].The assays were performed according to the manufacturers' instructions.

STATISTICAL ANALYSIS

Data were analyzed using Statistical Package for Social Science version 20 (IBM, SPSS Statistics 20), and graphs were generated with the help of GraphPad Prism 5 and Microsoft Excel. The results were considered significant if $p < 0.05$.

Ethical clearance was obtained from Institutional Ethics Review

Committee (IERC). All healthy volunteers were enrolled and a written informed consent was taken. The proforma included, name, age, sex, dietary habit, personal history of disease (if any), smoking habit, drinking habit, socioeconomic status and occupation.

RESULT

The study had 240 participants between the age groups of 20-65 years. The diabetic group included 80 males and 40 females whereas the control group included 78 males and 42 females. In the controls, the mean HbA1c level was 5.58 % and in the diabetic Patients, mean HbA1c level was 9.23 % which was shown in Table 1. The HbA1c distribution in both the study groups is shown in (Fig 1).

Table 1: - Comparison of HbA1c, HOMA IR among Healthy Control and Type 2 Diabetic Subjects

Variables	Healthy individual (Controls)	Diabetic patients (Cases)	p-Value
Fasting Glucose (mg/dl)	88.21±9.28	196.11±70.12	<.001**
Post Prandial Glucose (mg/dl)	125.33±10.45	265.94±88.81	<.001**
HbA1c (%)	5.58 ± 0.45	9.23 ± 1.66	<.001**
HOMA IR	1.18±0.42	12.96±6.95	<.001**

**: Significant at 1% level of significance
*P-Value <.001 considered as statistically significant

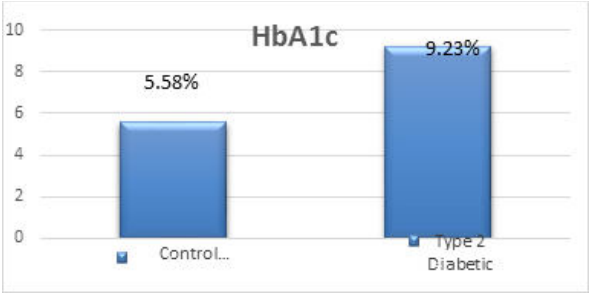

Fig 1: - HbA1c level of Control Group and Type 2 Diabetes.

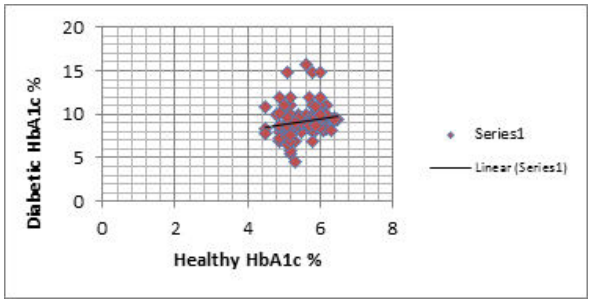

Fig 2: - Scatter plot showing Correlation values between healthy HbA1c Group & Diabetic Type II HbA1c Group

DISCUSSION

Diabetes is a group of metabolic disorders characterized by a chronic hyper glycemic condition resulting from insufficient action of insulins and it is the most common cause of macrovascular and microvascular complications, posing a huge international health burden[10]. The form of diabetes, which accounts for; 90–95% of those with diabetes, previously referred to as non–insulin dependent diabetes, type 2 diabetes, or adult-onset diabetes, encompasses individuals who have insulin resistance and usually have relative insulin deficiency[11].

The aim of our study was to find an association between Glucose, HbA1c and HOMA IR in healthy control and type 2 diabetic subjects. Ethical clearance was granted by the scientific and ethical committee of the institution. The study was conducted in Index Medical College, Hospital & Research Center (IMCHRC), Indore; patients were selected from medicine OPD. Consent was taken before sample collection from the patients. The present study included total 240 subjects, a control group of 120 subjects and study group of 120 subjects diagnosed with Type 2 Diabetes mellitus.

In our study, out of 120 subjects in each group, there were 42 females and 78 males in healthy group and 40 females with 80 males in diabetic group. In present study, we estimated BMI of both control and type 2 diabetes mellitus patients. We found significantly increased BMI level in diabetes group as compare to control healthy group. ($p \leq 0.001$)

In Present study, we estimated Fasting & Post Prandial glucose. We found FBS and PPBS level were significantly increased in Diabetic groups as compared to control group ($p \leq 0.001$) (Table 1). In Present study, mean values of HbA1c were 5.58 % and 9.23 % in healthy individual and type-2 diabetic respectively. They were found to be significantly increased in diabetic group as compared to control subjects (Fig.1) ($p \leq 0.001$). This is in accordance with Dr. Anand[12] Haddadinezhad S[13]. Dr. anand et al 2017 found increased HbA1c level is diabetes patients compared to healthy group. Haddadinezhad S et al (2010) showed that increasing of HbA1c has shown more dependency with postprandial plasma glucose as compared to with fasting plasma glucose.

In Present study, the mean value of HOMA IR in healthy control group and type 2 diabetic group were 1.18±0.42 and 12.96 ±6.95 respectively. Our study was concurrent with the study of Srihardyastutie[14] akira katsuki[15].Srihardyastutie et al 2014 found Insulin resistance (HOMAIR) were significantly higher in Type 2 Diabetes Mellitus than prediabetes and healthy control. akira katsuki et al 2000 found in their study that HOMA-IR may constitute a useful method not only for diagnosing insulin resistance, but also for follow-up during the treatment of patients with type 2 diabetes. Insulin is a peptide hormone secreted by β-cell of the pancreatic islet of Langerhans for maintaining normal blood glucose level by facilitating cellular glucose uptake and the other function of insulin prevents uncontrolled hydrolysis of triglyceride and limits gluconeogenesis, thereby maintaining normal fasting blood glucose level. An increasing of serum glucose will induce pancreatic β-cell to increase insulin secretion for maintaining the homeostasis of normal blood glucose. The condition was known as compensatory hyperinsulinemia condition[16].

In the present study, the associations of FPG and HbA1c with incident type 2 diabetes was indicated and there is also Positive correlation between HbA1c with HOMA IR in diagnosed type 2 diabetes mellitus and HOMA IR level can be improved with treatment of diabetes as shown in some pervious study. (Fig 2)

CONCLUSION

Fasting Glucose was most strongly associated with incident diabetes, followed by postprandial glucose level, HbA1c and HOMA-IR. and One of the most widely used validation measures for IR is the HOMA-IR index, which uses the determination of fasting insulin and glucose. The strong association of fasting glucose compared with 2hPG and HbA1c is in line with Fasting glucose being the most common marker for the diagnosis of type 2 diabetes. These results have potential implications for estimating the various diabetic complication. HbA1c and specially insulin levels were associated with metabolic syndrome criteria, and insulin resistance. Insulin could also help for better information in subjects prone to arise metabolic syndrome.

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COMPETING INTERESTS

The authors declare that we have no conflict of interests to declare.

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