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Pancreatic Beta-cell function and degree of Insulin resistance among newly detected type 2 diabetics and their correlation with anthropometric, glucose and lipid parameters: An Observational cross-sectional study

Avijit Saha

Associate Professor, Department of General Medicine, Malda Medical College and Hospital, Malda, West Bengal, India

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ABSTRACT

Background: Insulin resistance is a major cause for developing type 2 diabetes. However, simultaneously pancreatic beta-cell dysfunction must coexist in for clinical occurrence of type 2 diabetes (T2D). Hence, knowledge regarding residual beta-cell function and degree of insulin resistance is required while treating type 2 diabetic patients. **Aims and Objectives:** The present study was done to estimate degree of insulin resistance (homeostatic model assessment-insulin resistance - HOMA-IR) and pancreatic beta-cell functional capacity (HOMA-B%) among newly detected type 2 diabetics and correlation of these with anthropometric, glucose, and lipid parameters. **Materials and Methods:** This was an observational cross-sectional study conducted in 100 newly diagnosed type 2 diabetic patients. Detailed anthropometric and clinical examination were carried out. Venous blood samples were drawn for fasting plasma glucose, c-peptide, fasting insulin level, hemoglobin A1C (HbA1C), lipid profile, and postprandial glucose. HOMA-IR and HOMA-B% were calculated using HOMA 2 calculator and correlations were calculated between the study variables. **Results:** The mean age of the study population was 45.55 ± 11.64 years and 58% of study participants were male. The mean HOMA-IR and HOMA-B% were 2.55 ± 1.75 and 40.67 ± 23.55%, respectively. HOMA-IR positively correlated with abdominal circumference, triglyceride to HDLc ratio, and negatively correlated with HDLc. There were statistically significant negative correlations between HOMA-B% and fasting glucose($r = -0.48$, $P < 0.001$), 2 hr post prandial glucose ($r = -0.37$, $P < 0.001$ and HbA1C ($r = -0.24$, $P = 0.01$). **Conclusion:** This study found more reduced beta-cell function compared to reduced insulin sensitivity in new T2D mellitus patients. Hence, this kind of functional assessment needs to be done while selecting appropriate anti-diabetic drugs for a particular patient.

Key words: Type 2 diabetes mellitus; Homeostatic model assessment-insulin resistance; Homeostatic model assessment-B%

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INTRODUCTION

It has been known that type 2 diabetes (T2D) is not a homogeneous disease.[1] It can be hepatogenic or pancreatogenic or obesogenic or multifactorial. Now, eight to 12 different pathophysiological defects that are contributing to blood hyperglycemia in T2D were identified.[2] Now, it has been shown that kidney, brain, gut hormones, and gut microbiota all have contribution in the development of T2D.[3,4] At the same time, same of the recent studies proposed that incretin defect may be the result of chronic hyperglycemia and can be improved, once euglycemia is achieved.[5,6] Hence, we proposed that pancreatic beta-cell secretary defect and insulin resistance continue to be the

Address for Correspondence:
Dr. Avijit Saha, Malda Medical College Doctor's Quarter, Type 1, Flat No. 4B, Malda town, Malda - 732 101, West Bengal, India.
Mobile: +91-9883026884. **E-mail:** sahaavijit46@gmail.com

major determinant of T2D. Whatever forms of diabetes, it is, basically a simple imbalance between insulin secretion and insulin resistance. While European Association for the Study of Diabetes/American Diabetes Association (ADA) considered various comorbidities, while selecting various anti-diabetic medications, they did not mention the consideration of these basis endocrinal defects of T2D. What is the rationality behind giving a diabetic patient insulin secretagouge without knowing his or her beta-cell reserve? Pancreatic beta-cell reserve and degree of insulin resistance are not the same between a diabetic patient who is older in age, overweight, having long-standing diabetes with multiple comorbidities, and a diabetic patient who is younger, not overweight, recently been diagnosed and without any comorbidities.[7] Hence, knowledge about these parameters is of paramount important while treating type 2 diabetic patients. As hyperinsulinemic euglycemic clamp method is considered, as the gold standard for direct assessment of insulin resistance and insulin disposition index is a sensitive parameter of pancreatic beta-cell function.[8] However, these methods are difficult to implement in day-to-day clinical practice. There are several indirect ways to calculate these parameters. These are homeostatic model assessment (HOMA) model, quantitative insulin sensitivity check index, McAuley index, and triglyceride-glucose index. The values of these indices are consistent with the values derived from clamp-based studies.[9] In 1998, Levy et al., published an updated HOMA model (HOMA-2), where after putting fasting plasma glucose (FPG) and fasting plasma insulin levels, we can get individual's beta-cell function in the form of HOMA-B% and degree of insulin resistance in the form of HOMA-IR.[10] Wasana et al., in their study, calculated HOMA-IR, HOMA-B% values among newly diagnosed diabetic patients.[11] There are only few clinical studies done on this field particularly in Indian setting. In the previously published studies, no one had tried to find out clinical surrogate markers of these parameters.Hence,this present study was carried out to assess these parameters in diabetic patients at the time of diabetes diagnosis.

Aims and objectives

So, the present study was done to estimate the degree of insulin resistance in the form of HOMA-IR & pancreatic beta cell functional capacity in the form of HOMA-B% in newly detected treatment naïve type 2 diabetic patients and to estimate their correlation with anthropometric, glucose and lipid parameters.

MATERIALS AND METHODS

This was an observational cross-sectional single center study done in the department of General Medicine of Malda Medical College and Hospital, Malda, West Bengal from March 2020 to December 2021. This study included patients who had recently been diagnosed with T2D (as per ADA criteria) and came to our clinic for initiation of drug therapy. Those patients having other types of diabetes such as type 1, gestational diabetes mellitus, secondary diabetes and those presented with concurrent infection, other illnesses, or were in metabolic decomposition state were excluded from the study.

After detail explanation of study procedure, informed consent was taken from each participant and Institutional Ethical Clearance was obtained for the study. Details history, anthropometric measurements, and clinical examination were carried out. Body height was measured with a portable stadiometer. Weight was measured using a digital weighing scale. Both height and weight measurements were taken in light clothing without shoes. WC was measured as the midpoint circumference between the iliac crest and the lowest rib and BMI was calculated. Then, blood samples were taken far estimation of FPG, fasting plasma insulin, C-peptide, lipid profile, 2 h post prandial glucose (2 h PPG), and hemoglobin A1C (HbA1c). HOMA-2 calculator was used from where the HOMA-B% and HOMA-IR values were obtained. HOMA-IR value more than equal to 2.5 was considered as presence of insulin resistant and HOMA-B% ≤50% was considered as poor pancreatic beta-cell reserve.[12]

Sample size

Sample size was calculated assuming proportion of Assessment of Pancreatic beta-cell function and degree of insulin resistance as 40.27% as per the study by Basukala et al.[13] The other parameters considered for sample size calculation were 5% absolute precision and 95% confidence level. Based on the previous hospital records, the approximate number of potential eligible subjects to be attending the study setting during the data collection period were considered as 128. Hence, a finite population correction was applied for 128. The following formula was used for sample size as per the study by Daniel et al.[14]

$$n' = \frac{NZ^2P(1-P)}{d^2(N-1)+Z^2P(1-P)}$$

Where n=Sample size
N=Population size=128
Z=Z statistic for a level of confidence level=1.960
P=Expected prevalence/proportion of outcome=0.4028
d=Precision=0.05

The required sample size as per the above-mentioned calculation was 95. To account for a non-participation rate about 5%, another 5, subjects were added to the sample size. Hence, the final required sample size was 100.

Statistical methods

Assessment of pancreatic beta-cell function and degree of insulin resistance was considered as primary outcome of interest. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. For normally distributed quantitative parameters, the mean values were compared between study groups using ANOVA (>2 groups). Categorical outcomes were compared between study groups using Chi-square test. Association between quantitative explanatory and outcome variables was assessed by calculating Pearson correlation coefficient and the data were represented in a scatter diagram. P<0.05 was considered statistically significant. Data were analyzed using co Guide software, V.1.0.[15]

RESULTS

A total of 100 newly diagnosed treatment naive type 2 diabetic patients were studied. The mean age of the study population was 45.55±11.64 years. Among the study population, 58% comprised males. The mean BMI calculated was 25.93±4.39 kg/m^2. This falls under obese category as per Asian-Indian cutoff. The positive family history of diabetes was present in 33% of study population. The mean WCs in male and female participants were 90.16±11.63 cm and 89.91±10.25 cm, respectively. Hence, the study subjects were on average abdominally obese. The clinical and biochemical characteristics of study subjects are summarized in Table 1.The mean values of fasting plasma insulin and fasting c-peptide were 7.09±5.49 µIU/mL and 2.41±1.01 ng/mL, respectively. The mean values of HOMA-B% and HOMA-IR were 40.67±23.55 and 2.22±1.75, respectively. The pancreatic beta-cell functional capacity in the form of HOMA-B% values in the study population is as follows – 39% of study population had HOMA-B% values below 30% and 32% participants had values between 30% and 50%. Overall 49% of study population had HOMA-IR values below 2.5 which mean that they did not have significant amount of insulin resistance at baseline. The remaining 51% patients had a significant amount of insulin resistance at the time of disease diagnosis as their HOMA-IR values were more than equal to 2.5. The Pearson correlation coefficients between HOMA-B% and BMI, abdominal circumference, FPG, 2 h PPG, HbA1c, low-density lipoprotein cholesterol (LDLc), high-density lipoprotein cholesterol (HDLc), and triglyceride are represented in Table 2. Accordingly, HOMA-B% showed significant negative correlation with all plasma glucose indices, namely, FPG, 2 h PPG, and HbA1c (Figure 1). Whereas HOMA-B% was negatively correlated with all lipid parameters, but these did not attain statistical significance. The Pearson correlation coefficients between HOMA-IR and BMI, abdominal circumference, FPG, 2hrPPG, HbA1c, LDLc, HDLc, triglyceride, and triglyceride/HDLc ratio are represented in Table 3. HOMA-IR significantly positively correlated with abdominal circumference, but there was no significant correlation found with BMI. Among the lipid parameters, HOMA-IR significantly and positively correlated with triglyceride to HDLc ratio and negatively correlated with HDLc (Figure 2). The most common pattern of lipid profile abnormality found in the study cohort was high triglyceride (68%cases) followed by combined low HDLc and high triglyceride (51% cases). Isolated high LDLc was found in 23% cases.

Table 1: Clinical and laboratory parameters in the study population (n=100)

Variables	Mean±SD	95% confidence interval
Age (years)	45.55±11.64	43.27–47.83
Gender (M:F)	58:42	-
BMI (kg/m^2)	25.93±4.39	25.07–26.79
Waist circumference (male) (cm)	90.16±11.63	87.11–93.22
Waist circumference (female)	89.91±10.25	86.72 – 93.10
FPG (mg/dL)	193.77±79.96	178.10–209.44
2 h PPG (mg/dL)	296.55±114.07	274.19–318.91
HbA1c (%)	8.40±1.91	8.03–8.77
TC (mg/dL)	177.25±40.28	169.35–185.15
LDLc (mg/dL)	109.52±30.14	103.61–115.43
HDLc (mg/dL)	39.26±6.73	37.94–40.58
Triglycerides (mg/dl)	205.44±107.72	184.33–226.55
TG/HDLc ratio	4.5±3.9	5.2–6.7
Fasting plasma insulin (µIU/mL)	7.09±5.49	6.01–8.16
Fasting C-peptide (ng/mL)	2.41±1.01	2.22–2.61
HOMA-IR	2.55±1.75	2.21–2.89
HOMA-B%	40.67±23.55	36.05–45.28

BMI: Body mass index, FPG: Fasting plasma glucose, 2 h PPG: 2 h post prandial glucose, HbA1c: Glycated hemoglobin, LDLc: Low-density lipoprotein cholesterol, HDLc: High-density lipoprotein cholesterol. TG: Triglycerides. HOMA-B%: Homeostatic model assessment for beta-cell function, HOMA-IR: Homeostatic model assessment for insulin resistance, TC: Total cholesterol

Table 2: Correlation between HOMA-B% and various parameters in the study population (n=100)

Parameters	Pearson correlation	P value
BMI	0.054	0.669
Abdominal circumference	0.409	<0.001
FPG	−0.487	<0.001
2 h PPG	−0.373	<0.001
HbA1c	−0.246	0.013
LDLc	−0.043	0.672
HDLc	−0.096	0.340
TG	−0.050	0.621

BMI: Body mass index, FPG: Fasting plasma glucose, 2 h PPG: 2 h post prandial glucose, HbA1c: Glycated hemoglobin, LDLc: Low density lipoprotein cholesterol, HDLc: High-density lipoprotein cholesterol. TG: Triglycerides. HOMA-B%: Homeostatic model assessment for beta-cell function

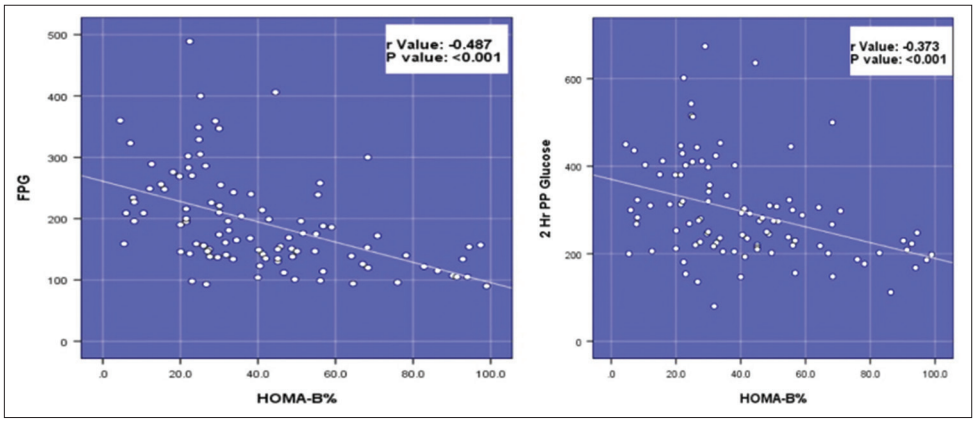

Figure 1: Scatter plots between homeostatic model assessment for beta-cell function(HOMA-B%) and Fasting plasma glucose, 2 h post prandial glucose

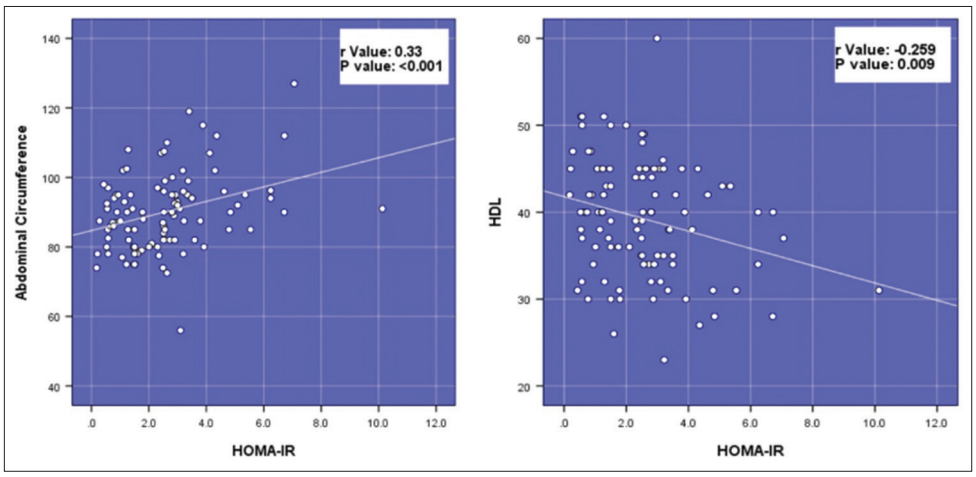

Figure 2: Scatter plots between homeostatic model assessment for insulin resistance(HOMA-IR) and abdominal circumference and high-density lipoprotein cholesterol

Table 3: Correlation between HOMA-IR and various parameters in the study population (n=100)

Parameters	Pearson Correlation	P value
BMI	0.142	0.281
Abdominal circumference	0.33	<0.001
FPG	−0.116	0.249
2 h PPG	−0.118	0.240
HbA1c	−0.103	0.308
LDLc	−0.119	0.237
HDLc	−0.259	0.009
TG	0.053	0.604
TG/HDLc ratio	0.096	0.021

BMI: Body mass index, FPG: Fasting plasma glucose, 2 h PPG: 2 h post prandial glucose, HbA1c: Glycated hemoglobin, LDLc: Low-density lipoprotein cholesterol, HDLc: High-density lipoprotein cholesterol. TG: Triglycerides.
HOMA-IR: Homeostatic model assessment for insulin resistance

DISCUSSION

To the best of our knowledge, the present study is one of the few studies of South Asian population attempting to assess baseline pancreatic beta-cell function and degree of insulin resistance in type 2 diabetic patients at the time of diagnosis. The present study was also searched for surrogate markers that would clinically predict the extent of defects of these parameters of T2D. The present study demonstrated significant positive correlation of WC with HOMA-IR, but BMI did not meaningfully correlated with HOMA-IR. As a traditional marker of obesity, BMI has many limitations to predict cardiometabolic risk because having same BMI, persons may have different visceral adiposity. Hence, WC or waist-hip ratio could be a better predictor and clinical marker of insulin resistance. The excess visceral fat depot that is the source of different inflammatory cytokines that impair insulin sensitivity on target organs. That could be the probable explanation of this association.[16] The mean values of fasting plasma insulin and fasting c-peptide were 7.09±5.49 μIU/mL and 2.41±1.01 ng/mL, respectively. These values are low in our study cohort compared to study done by Chen et al.,[17] who reported a mean fasting insulin level of 10.37 μIU/mL in their study population. Wasana et al.,[11] did a similar kind of study among newly detected type 2 diabetic patients and found mean fasting plasma

insulin level of 18.15±10.81 μIU/mL. This could be due to low beta-cell reserve in our ethnicity as a result of low birth weight, prematurity, and malnutrition.[18] The mean value of HOMA-IR was found to be 2.22±1.75 in the present study. Wasana et al.,[11] reported mean HOMA-IR 5.51±3.85 in their study cohort. In the present study, HOMA-B% showed significant negative correlation with all plasma glucose indices, namely, FPG, 2 h PPG, and HbA1c. That means with the increasing HOMA-B values that there were decreasing values of glucose in plasma. Hence, when beta-cell fails to compensate in response to significant amount of insulin resistance, then clinically overt T2D manifests. Surprisingly, we did not find any significant correlation of HOMA-IR with any of the glucose parameters in our diabetic study population. In our study, among the lipid parameters, HOMA-IR was significantly positively correlated with triglyceride to HDLc ratio and negatively correlated with HDLc. The previous studies have also proved that triglycerides (TG)/HDL-C ratio can be a good surrogate marker for individual's degree of insulin resistance.[19,20] The potential mechanism behind this association could be free fatty acids, triacylglycerol derived from triglycerides interfere with insulin signaling at target tissues.[21] The presence of dyslipidemia was quite high in the study participants. The most common pattern of lipid profile abnormality was high triglyceride (68% cases) followed by combined low HDLc and high triglyceride (51% cases). Isolated high LDLc was found in 23% cases. The association of combined high triglycerides with low HDLc levels and T2D is well accepted in several studies. In concurrence with this, the ADA recommends that all adults should be screened for diabetes mellitus if they have a TG levels >250 mg/dL and/or HDL-C levels <35 mg/d.[22] Moreover, Giannini et al.,[23] proposed that "the TG to HDL-C ratio is correlated with insulin sensitivity and may be used to identify subjects with high degree of insulin resistance and cardiometabolic risk." Dyslipidemia is considered as an independent risk factor for the development of T2D mellitus (T2DM). Chen et al.,[24] have found that subjects with hyperlipidemia were more than 3 times higher risk of having T2DM compared with participants with normolipidemia. Although it has been said that dyslipidemia has a negative impact on β-cells functions in diabetic individuals, the solid evidence of different lipid parameters (TG, total cholesterol, HDL-C, and LDL-C) on insulin sensitivity and β-cell function is still unclear. The present study also demonstrated that in more than two-third study population pancreatic beta-cell function had reduced to <50% by the time of diagnosis and more than half of the study population had significant degree of insulin resistance.

The main strength of this study is that the inclusion of newly diagnosed patients who had not been initiated with any glucose and lipid lowering agents that could have modifying effects on pancreatic beta-cell function and insulin resistance.

The study findings have to be tested prospectively in a larger cohort of patients with different ethnicity through well-planned randomized clinical trials.

Limitations of the study

The present study had certain limitations like it was done in a single tertiary care center, so generalizability of study findings to other parts of India was not ascertained. Similarly, we had done this assessment by indirect method and had not followed up these patients with serial measurement of these parameters over the course of disease progression. HOMA-2 calculator has its own limitations like it is unreliable in the presence of very high blood glucose value and in presence of poor beta-cell function.[25]

CONCLUSION

This study demonstrated that every diabetic patient is different from each other in terms of their residual pancreatic beta-cell functional capacity and degree of insulin resistance. Our study found more reduced beta-cell function compared to reduced insulin sensitivity in new T2DM patients. Hence, this kind of functional assessment needs to be done for selection of appropriate anti-diabetic drugs for a particular pt.Waist circumferenceand TG/HDL-C ratio were found to be good clinical surrogate markers of insulin resistance. Findings of this study will enable clinicians to adopt most optimal treatment plan for patients with newly diagnosed T2DM based on their age, BMI, WC, HDLc level, and TG/HDL ratio and HOMA 2 calculator derived HOMA-B% and HOMA-IR parameters and without opting for any expensive testing.

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AS- Study concept & design,acquisition & analysis of data,drafting & critical revision of the manuscript.

Work attributed to:
Malda Medical College and Hospital, Diabetes Clinic, Medicine OPD, Malda, West Bengal.

Orcid ID:
Avijit Saha - https://orcid.org/0000-0002-8956-0834

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