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ORIGINAL ARTICLE

Correlation between obesity and glycated hemoglobin in patients with type II diabetes mellitus

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ABSTRACT:

Background: Obesity and Type 2 diabetes (T2DM) together are becoming a fatal mix. While the precise causes of T2DM are still mostly unknown, a number of factors are thought to be involved. The present study evaluated correlation between obesity and glycated hemoglobin in type II diabetes patients. **Materials & Methods:** 110 type II diabetes mellitus patients of both genders were divided into two categories: Normal BMI (18.0–22.9 kg/m2), Overweight (23.0–24.9 kg/m2), and Obesity (>25 kg/m2). Glucose oxidase was used to measure fasting plasma glucose, and Latex agglutination inhibition assay was used to measure HbA1c. **Results:** Out of 110 patients, males were 68 and females were 42. There were 34 patients with BMI between 18.5-25 kg/m^2, 30 with 25-30 kg/m^2 and 46 with 25-30 kg/m^2 BMI. The difference was significant (P< 0.05). The mean FBG was 152.4 mg/dl, 147.6 mg/dl and 170.2 mg/dl, SBP was 138.4 mm Hg, 129.4 mm Hg and 134.6 mm Hg, DBP was 90.2 mm Hg, 74.2 mm Hg and 82.2 mm Hg, WC was 82.4 cm, 96.5 cm and 101.3 cm and HbA1C was 6.1%, 8.4 % and 8.7% in normal, overweight and obese patients respectively. The difference was significant (P< 0.05). **Conclusion:** The prevalence of obesity was greater in persons with diabetes. Among diabetes subjects with medium BMI who were also overweight or obese, dysglycemia was elevated.

Key words: Diabetic, obese, dysglycemia

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INTRODUCTION

Obesity and Type 2 diabetes (T2DM) together are becoming a fatal mix. While the precise causes of T2DM are still mostly unknown, a number of factors are thought to be involved. Obesity and type 2 diabetes are closely related, according to earlier research.Individuals who are overweight or obese have an increased risk of type 2 diabetes. According to recent data, nearly 1.9 billion adults aged 18 and above were overweight. More than 600 million of them were fat.[1]

The etiology of type 2 diabetes and associated macrovascular consequences is largely dependent on obesity. Nevertheless, due to their physiologically unfavorable profile, which includes hyperinsulinemia, insulin resistance, and hypertriglyceridemia, certain people who are normal weight have a significant risk of developing T2D and cardiovascular disease. Because of this, having a high body mass index (BMI) is not required for the development of these problems, indicating that the underlying mechanisms causing T2D's cardiovascular consequences are complex. The relative contribution of obesity to the cardiovascular risk of patients who already have a higher risk of cardiovascular problems due to T2D may be shown by epidemiologic study on cardiovascular risk variables among patients with T2D and different BMI ranges.[2]

In obese people who eventually develop type 2 diabetes, insulin resistance and insulin secretion both start early.4 Changes in diet, nutrition, and lifestyle may be the cause of the rise in T2DM incidence in developed nations. In contrast to the population in the West, diabetes is becoming more common in India, despite lower rates of overweight and obesity. This demonstrates that T2DM can develop in Indians at significantly lower body mass indices (BMIs).[3] The present study evaluated correlation between obesity and glycated hemoglobin in type II diabetes patients.

MATERIALS & METHODS

The present study consisted of 110 type II diabetes mellitus patients of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. Biochemical indicators and a thorough history were evaluated. The length of diabetes and any family history of the condition were noted. Weight, height, and BMI (kg/m2) are examples of anthropometric metrics that have been calculated. Measured was the waist circumference (WC). Individuals were divided into two categories: Normal BMI (18.0–22.9 kg/m2), Overweight (23.0–24.9 kg/m2), and Obesity (>25 kg/m2). Glucose oxidase was used to measure fasting plasma glucose, and Latex agglutination inhibition assay was used to measure HbA1c. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 110		
Gender	**Males**	**Females**
Number	68	42

Table I shows that out of 110 patients, males were 68 and females were 42.

Table II Distribution of patients based on BMI

BMI	Number	P value
18.5-25 kg/m^2	34	0.91
25-30 kg/m^2	30	
>30 kg/m^2	46	

Table II shows that there were 34 patients with BMI between 18.5-25 kg/m^2, 30 with 25-30 kg/m^2 and 46 with 25-30 kg/m^2 BMI. The difference was significant (P< 0.05).

Graph I Distribution of patients based on BMI

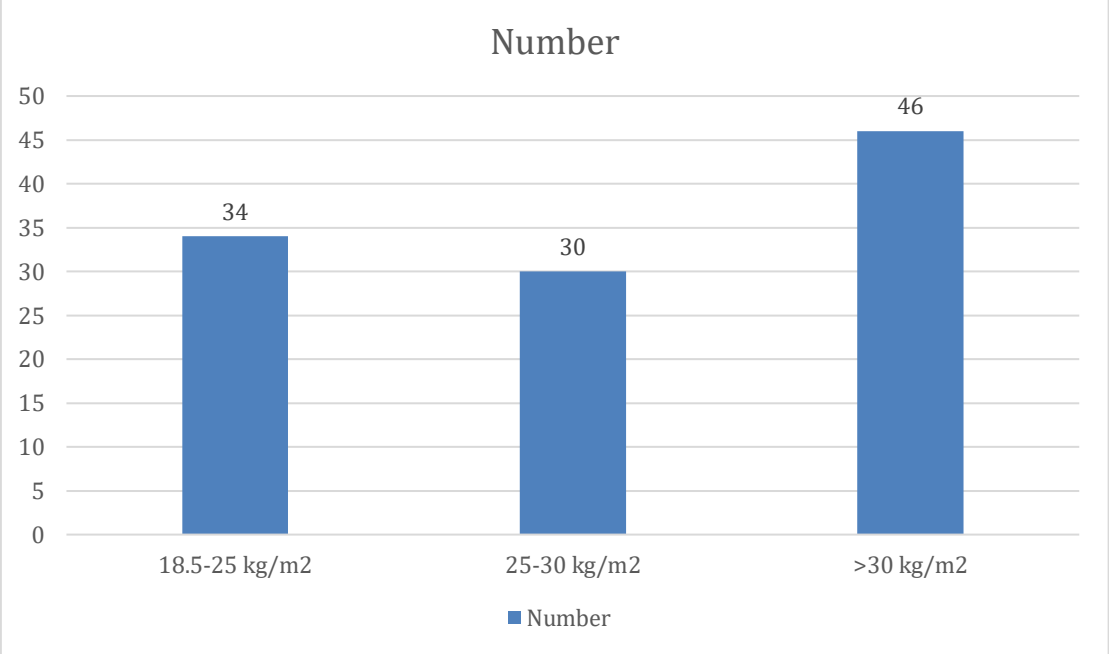

Table III Assessment of parameters

Parameters	Normal	Overweight	Obese	P value
FBG (mg/dl)	152.4	147.6	170.2	0.05
SBP (mm Hg)	138.4	129.4	134.6	0.94
DBP (mm Hg)	90.2	74.2	82.2	0.42
WC (cm)	82.4	96.5	101.3	0.05
HbA1C (%)	6.1	8.4	8.7	0.01

Table III shows that mean FBG was 152.4 mg/dl, 147.6 mg/dl and 170.2 mg/dl, SBP was 138.4 mm Hg, 129.4 mm Hg and 134.6 mm Hg, DBP was 90.2 mm Hg, 74.2 mm Hg and 82.2 mm Hg, WC was 82.4 cm, 96.5 cm and 101.3 cm and HbA1C was 6.1%, 8.4 % and 8.7% in normal, overweight and obese patients respectively. The difference was significant (P< 0.05).

DISCUSSION

The most prevalent metabolic disease, diabetes mellitus, is characterized by a number of hormone-induced metabolic abnormalities as well as long-term complications. Over the past 20 years, there has been a significant increase in the global prevalence of diabetes mellitus, with an estimated 30 million cases in 1985 and 415 million cases in 2017.[4] A condition of excess adipose tissue mass is obesity. Nevertheless, this strategy increases adipose energy storage and has negative health effects in the context of a sedentary lifestyle, abundant eating, and significant genetic endowment.[5]

Obesity is characterized by hyperinsulinemia and insulin resistance, which rise with weight gain and fall with weight loss. Unknown is the precise etiology of microvascular problems in diabetes mellitus.[6] The onset and progression of endothelial inflammation are

influenced by various factors, including oxidative stress-activated Renin Angiotensin System (RAS), hyperglycemia, Advanced Glycosylation End-products (AGE), and oxidized low-density lipoproteins. These factors ultimately result in diabetic vascular problems.[7,8] The present study evaluated correlation between obesity and glycated hemoglobin in type II diabetes patients.

We found that out of 94 patients, males were 54 and females were 40. Sheth et al[9] found that in both T2DM and non-diabetic patients, dyslipidemia and obesity were associated with glycated hemoglobin (HbA1c). With precise anthropometric measurements, the current study involved 931 participants from urban Western India, 430 of whom had diabetes and 501 of whom did not. HbA1c and lipid indices such as TC, TG, HDL-C, LDL-C, and non-HDL-C were examined for each individual. All research participants had dyslipidemia, as well as central and peripheral obesity, with percentages of 50.27%, 75%, and 598.33%, respectively. Furthermore, in T2DM and non-diabetic patients, hyper-non-HDL-C was found in 23.49% and 22.56% of cases, respectively. Hyper-TC, hyper-LDL-C, and hyper-non-HDL-C were found to have significant linear correlations with HbA1c in individuals with type 2 diabetes and non-diabetic control participants, respectively. Both peripherally and centrally obese dyslipidemic participants demonstrated a strong correlation between HbA1c in T2DM and control.

We observed that out of 110 patients, males were 68 and females were 42. There were 34 patients with BMI between 18.5-25 kg/m^2, 30 with 25-30 kg/m^2 and 46 with 25-30 kg/m^2 BMI. Huh et al[10] assessed the direct and indirect effects of BMI on GA/A1c ratio, structural equation modeling (SEM) was performed. GA/A1c ratio was set as a dependent variable, BMI was used as the independent variable, and homeostasis model assessment-pancreatic beta-cell function (HOMA-β), homeostasis model assessment-insulin resistance (HOMA-IR), glucose level were used as mediator variables. The estimates of a direct effect of BMI on GA/A1c to be the strongest in NGT and weakest in T2D (−0.375 in NGT, −0.244 in prediabetes, and −0.189 in T2D). Conversely, the indirect effect of BMI on GA/A1c exerted through HOMA-β and HOMA-IR was not statistically significant in NGT group, but significant in prediabetes and T2D groups (0.089 in prediabetes, −0.003 in T2D). It was found that HOMA-β or HOMA-IR indirectly influences GA/A1c in T2D and prediabetes group through affecting fasting and postprandial glucose level. The relationship between GA/A1c and BMI is due to the direct effect of BMI on GA/A1c in NGT group, while in T2D and prediabetes groups, this association is mostly a result of BMI influencing blood glucose through insulin resistance or secretion.

We found that the mean FBG was 152.4 mg/dl, 147.6 mg/dl and 170.2 mg/dl, SBP was 138.4 mm Hg, 129.4 mm Hg and 134.6 mm Hg, DBP was 90.2 mm Hg, 74.2 mm Hg and 82.2 mm Hg, WC was 82.4 cm, 96.5 cm and 101.3 cm and HbA1C was 6.1%, 8.4 % and 8.7% in normal, overweight and obese patients respectively. Prabhavati[11] tested whether HbA1c can serve as a marker of circulating lipids among Type 2 diabetic patients. The sera of 130 Type 2 diabetic patients was analyzed for fasting blood sugar (FBS), HbA1c and lipid profile consisting of total cholesterol (TC), triglycerides (TG), High-density Lipoprotein (HDL) cholesterol and LDL cholesterol. We divided the subjects based on their glycemic index into three groups; HbA1c< 6% as good, HbA1c>6% - <9% as poor and HbA1c>9% as worst glycemic control. The mean ± SD levels of HbA1c was significantly higher in females (8.598 ± 2.284 %) compared to males (7.323±2.18 %). Older patients had HbA1c, FBS and lipid profile levels similar to younger ones. HbA1c showed direct and significant correlations with cholesterol, TG and LDL. Univariate analysis showed that HbA1c was a good predictor of circulating lipid levels.

CONCLUSION

Authors found that the prevalence of obesity was greater in persons with diabetes. Among diabetes subjects with medium BMI who were also overweight or obese, dysglycemia was elevated.

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