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ORIGINAL RESEARCH

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

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ABSTRACT
Background: Numerous hormone-induced metabolic irregularities and long-term consequences define diabetes mellitus, the most common metabolic disorder. The present study assessed correlation between obesity and glycated hemoglobin in type II diabetes patients. **Materials & Methods:** 80 type II diabetes mellitus patients of both genders were selected. Weight, height, and BMI, waist circumference (WC), HbA1c and plasma glucose was determined. **Results:** Out of 80 patients, males were 56 and females were 24. There were 20 normal patients, 24 overweight and 36 obese. The difference was non-significant (P> 0.05). The mean WC was 80.4 cm, 94.5 cm and 102.5 cm, HbA1C was 6.7%, 8.1% and 8.9% in normal, overweight and obese patients respectively. FBG was 136.4 mg/dl, 142.8 mg/dl and 156.2 mg/dl. SBP was 134.2 mm Hg, 126.0 mm Hg and 136.4 mm Hg, DBP was 82.2 mm Hg, 86.4 mm Hg and 88.4 mm Hg in normal, overweight and obese patients respectively. The difference was significant (P< 0.05). **Conclusion:** Those with diabetes had higher obesity prevalence. Dysglycemia was higher in patients with diabetes who had a medium BMI and was overweight or obese.
Key words: Diabetic, obese, dysglycemia

INTRODUCTION

Numerous hormone-induced metabolic irregularities and long-term consequences define diabetes mellitus, the most common metabolic disorder. Globally, the prevalence of diabetes mellitus has increased significantly over the last 20 years, from an estimated 30 million cases in 1985 to 415 million cases in 2017.[1] Obesity is a state of having too much adipose tissue mass. However, when combined with a sedentary lifestyle, a high intake of food, and a substantial genetic endowment, this approach raises the amount of adipose energy storage and has detrimental health implications.[2]

Obesity has a major role in the genesis of type 2 diabetes and its macrovascular complications. However, certain individuals who are normal weight have a high risk of developing type 2 diabetes and cardiovascular disease because of their physiologically unfavorable profile, which includes hyperinsulinemia, insulin resistance, and hypertriglyceridemia.[3] Consequently, the development of these issues does not seem to be correlated with a high body mass index (BMI), suggesting that the underlying mechanisms responsible for the cardiovascular repercussions of type 2 diabetes are intricate. An epidemiologic investigation on cardiovascular risk factors among patients with T2D and varying BMI ranges may demonstrate the relative contribution of obesity to the cardiovascular risk of patients who already have a higher risk of cardiovascular problems because of T2D.[4]

Insulin secretion and resistance begin early in obese individuals who subsequently develop type 2 diabetes. The increased occurrence of type 2 diabetes in developed countries could be attributed to dietary, nutritional, and lifestyle changes.[5] India's diabetes rate is rising relative to the West, even though the country's overweight and obesity rates are lower. This indicates that at much lower body mass indices (BMIs), T2DM might occur in Indians.[6] The present

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study assessed correlation between obesity and glycated hemoglobin in type II diabetes patients.

MATERIALS & METHODS

The present study consisted of 80 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. A detailed history and biochemical markers were assessed. Notable were the duration of the diabetes and any family history of the illness. Three examples of computed anthropometric measurements are weight, height, and BMI (kg/m2). The waist circumference (WC) was measured. A normal BMI range of 18.0–22.9 kg/m2, overweight range of 23.0–24.9 kg/m2, and obesity range of >25 kg/m2 were used to categorize the individuals. HbA1c was measured using the Latex Agglutination Inhibition Assay, whereas fasting plasma glucose was determined using glucose oxidase. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 80		
Gender	**Males**	**Females**
Number	56	24

Table I shows that out of 80 patients, males were 56 and females were 24.

Table II Assessment of BMI

BMI	Number	P value
Normal	20	0.61
Overweight	24	
Obese	36	

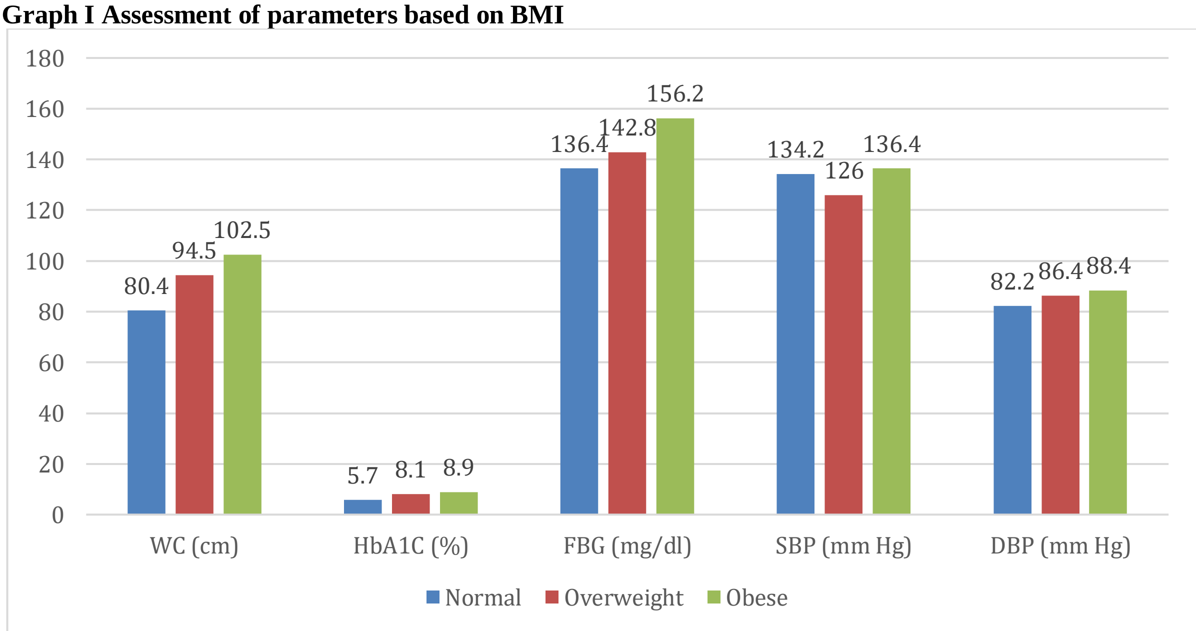
Table II shows that there were 20 normal patients, 24 overweight and 36 obese. The difference was non-significant (P> 0.05).

Table III Assessment of parameters based on BMI

Parameters	Normal	Overweight	Obese	P value
WC (cm)	80.4	94.5	102.5	0.05
HbA1C (%)	5.7	8.1	8.9	0.01
FBG (mg/dl)	136.4	142.8	156.2	0.05
SBP (mm Hg)	134.2	126.0	136.4	0.04
DBP (mm Hg)	82.2	86.4	88.4	0.52

Table III, graph I shows that mean WC was 80.4 cm, 94.5 cm and 102.5 cm, HbA1C was 6.7%, 8.1% and 8.9% in normal, overweight and obese patients respectively. FBG was 136.4 mg/dl, 142.8 mg/dl and 156.2 mg/dl. SBP was 134.2 mm Hg, 126.0 mm Hg and 136.4 mm Hg, DBP was 82.2 mm Hg, 86.4 mm Hg and 88.4 mm Hg in normal, overweight and obese patients respectively. The difference was significant (P< 0.05).

Graph I Assessment of parameters based on BMI



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DISCUSSION

Type 2 diabetes (T2DM) and obesity are increasingly associated with death. Although the exact origins of type 2 diabetes remain largely unknown, several factors are considered to be implicated.[7] Previous study has indicated a close relationship between type 2 diabetes and obesity. Obese or overweight people are more likely to develop type 2 diabetes. Nearly 1.9 billion persons over the age of 18 were overweight, according to current data.[8] Among them, about 600 million were obese. Two indicators of obesity that rise with weight gain and fall with weight loss, respectively, are hyperinsulinemia and insulin resistance.[9] It is unclear what specifically causes microvascular problems in people with diabetes mellitus. Many factors, including oxidized low-density lipoproteins, hyperglycemia, AGE (Advanced Glycosylation End-products), and the Renin Angiotensin System (RAS) triggered by oxidative stress, all affect the initiation and development of endothelial inflammation. Diabetic vascular complications are ultimately caused by these variables.[10-14] The present study assessed correlation between obesity and glycated hemoglobin in type II diabetes patients.

We found that out of 80 patients, males were 56 and females were 24. There were 20 normal patients, 24 overweight and 36 obese. Garg et al[15] evaluated the effect of obesity on SpO2 in a wide range of glycated hemoglobin (HbA1c) levels in ambulatory type 2 diabetic patients. A cohort of 60 subjects irrespective of diabetic status were recruited and clustered in group I (HbA1c <6.5) and group II (HbA1c ≥ 6.5) depending on HbA1c. Anthropometry and routine biochemical parameters were measured. HbA1c (%) were estimated by high performance liquid chromatography (HPLC) respectively. SpO2 (%) levels were measured by pulse oximetry. Blood concentration of HbA1c was <6.5 in 29 participants and ≥ 6.5 in 31 participants. Plasma fasting and post prandial glucose, HbA1c as well as Hb levels were significantly ($p<0.50$) higher in diabetics as compared to non- diabetics. Waist circumference (WC) ($r=-400$; $p=0.026$) and body mass index (BMI) ($r=-381$; $p=0.034$) showed a significant negative correlation with SpO2 in diabetic patients. On adjusting HbA1c in group II, SpO2 was found to independently and inversely associated with WC ($p=0.042$) and BMI ($p=0.049$).

We observed that the mean WC was 80.4 cm, 94.5 cm and 102.5 cm, HbA1C was 6.7%, 8.1% and 8.9% in normal, overweight and obese patients respectively. FBG was 136.4 mg/dl, 142.8 mg/dl and 156.2 mg/dl. SBP was 134.2 mm Hg, 126.0 mm Hg and 136.4 mm Hg, DBP was 82.2 mm Hg, 86.4 mm Hg and 88.4 mm Hg in normal, overweight and obese patients respectively. Luo et al[16] in their study a total of 3173 patients with T2DM participated in the study at the Metabolic Management Center (MMC), with anthropometric and biochemical measurements recorded. HbA1c was inversely associated with VFA (β −1.79, 95% CI −2.34~−1.24, $P < 0.001$). The fitted curve shows that VFA increased with the increase of HbA1c when it was less than 8.62%. When it was greater than 8.62%, VFA decreased as HbA1c increased. Using linear inflection point analysis, we found that its inflection point interval falls within 8.36%~8.88%. VFA was positively associated with HbA1c in individuals with T2DM. Furthermore, the relationship between the two variables was an inverted U-shaped association.

Sheth et al[17] discovered a correlation between glycated hemoglobin (HbA1c) and dyslipidemia in people with T2DM and those without diabetes. The current study included 931 people from urban Western India with accurate anthropometric data; 430 had diabetes and 501 did not. Each person's HbA1c as well as lipid indices including TC, TG, HDL-C, LDL-C, and non-HDL-C were assessed. Every research participant had a proportion of 50.27%, 75%, and 598.33% for central and peripheral obesity, respectively, along with dyslipidemia. Moreover, hyper-non-HDL-C was discovered in 23.49% and 22.56% of instances, respectively, among patients with T2DM and non-diabetic patients. In people with type 2 diabetes and non-diabetic control participants, it was discovered that hyper-TC, hyper-LDL-C, and hyper-non-HDL-C had strong linear associations with HbA1c, respectively. HbA1c in T2DM and control showed a substantial association in both peripherally and centrally obese dyslipidemic subjects.

CONCLUSION

The present study underscores the significant correlation between obesity and glycated hemoglobin (HbA1c) levels in patients with type II diabetes mellitus (T2DM). Our findings reveal that obese patients exhibit higher HbA1c, fasting blood glucose (FBG), and waist circumference (WC) compared to their normal and overweight counterparts. These results align with existing literature, indicating that obesity exacerbates glycemic control and increases cardiovascular risk in T2DM patients. Additionally, the study highlights the complexity of diabetes-related cardiovascular risks, which are not solely dependent on BMI but also involve metabolic and genetic factors. The data emphasize the need for tailored interventions focusing on weight management and metabolic control to mitigate the adverse health outcomes associated with T2DM. Further research is warranted to explore the intricate mechanisms linking obesity, insulin resistance, and cardiovascular complications in diabetic patients, especially in populations with varying BMI thresholds like those in India.

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