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ORIGINAL ARTICLE

Assessment of clinical and biochemical profiles of lean type 2 diabetes mellitus patients

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ABSTRACT:

Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by high blood sugar levels (hyperglycemia) resulting from insulin resistance and relative insulin deficiency. The present study was conducted to assess clinical and biochemical profiles of lean type 2 diabetes mellitus (T2DM) patients. **Materials & Methods:**84 type 2 diabetes mellitus patients with BMI less than 19 of both genders were selected. Anthropometric parameters – height, weight, waist, hip circumference, and waist-hip ratio, fasting and two hours postprandial blood glucose (BG) and HbA1c (glycosylated haemoglobin), lipid profile such as TG, HDL, LDL, TC, and VLDL were measured. **Results:** Out of 84 patients, males were 52 and females were 32. In males and females, mean height (cm) was 165.8 and 151.3, weight (kg) was 50.2 and 42.9, BMI (kg/m2) was 16.7 and 18.4, WC (cm) was 76.4 and 71.2, HC (cm) was 82.5 and 81.3, waist-hip ratio was 0.97 and 0.87, SBP (mm Hg) was 128.4 and 126.2, and DBP (mm Hg) was 76.2 and 74.8 respectively. The difference was significant (P< 0.05). The mean fasting BG (mg%) was 134.8, postprandial BG (mg%) was 201.4, HbA1c (%) was 7.6, TC (mg%) was 186.2, TG (mg%) was 145.2, LDL (mg%) was 108.4, HDL (mg%) was 49.2, and VLDL (mg%) was 32.6. **Conclusion:** Lean people with type 2 diabetes mellitus (T2DM) hasdyslipidemia, making the condition a unique clinical entity.

Keywords: diabetes mellitus, postprandial blood glucose, lipid profile

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by high blood sugar levels (hyperglycemia) resulting from insulin resistance and relative insulin deficiency. In T2DM, the body's cells become resistant to the effects of insulin, a hormone produced by the pancreas that helps regulate blood sugar levels.Insulin resistance means that cells are less responsive to insulin's signals, leading to impaired glucose uptake by tissues such as muscle and fat.[1]In addition to insulin resistance, T2DM is often associated with dysfunction of pancreatic beta cells, which are responsible for producing and secreting insulin.Over time, beta cells may become unable to produce enough insulin to overcome insulin resistance, resulting in relative insulin deficiency.[2]

Various studies in India have reported a prevalence of low body weight/lean (Body mass index, BMI.[3]Lean individuals with T2DM often present with a lower BMI (<25 kg/m^2) compared to the general population of T2DM patients.Despite their lower BMI, lean T2DM patients may exhibit central obesity, characterized by increased waist circumference and visceral adiposity.[4]They may have a family history of diabetes, suggesting a genetic predisposition to the disease.Lean T2DM patients may have a younger age of onset compared to overweight or obese individuals with T2DM.[5]The present study was conducted to assess clinical and biochemical profiles of lean type 2 diabetes mellitus (T2DM) patients.

MATERIALS & METHODS

The present study consisted of 84 type 2 diabetes mellitus patients with BMI less than 19of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. Anthropometric parameters – height, weight, waist, hip circumference, and waist-hip ratio were recorded. Glycemic status was assessed using fasting and two hours postprandial blood glucose (BG) and HbA1c (glycosylated haemoglobin).Lipd profile such as TG, HDL, LDL, TC, and VLDL were measured. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 84		
Gender	**Male**	**Female**
Number	52	32

Table I shows that out of 84 patients, males were 52 and females were 32.

Table II Assessment of clinical parameters

Parameters	Males	Females	P value
Height (cm)	165.8	151.3	0.05
Weight (kg)	50.2	42.9	0.03
BMI (kg/m2)	16.7	18.4	0.01
WC (cm)	76.4	71.2	0.04
HC (cm)	82.5	81.3	0.15
waist-hip ratio	0.97	0.87	0.19
SBP (mm Hg)	128.4	126.2	0.25
DBP (mm Hg)	76.2	74.8	0.82

Table II, graph I shows that in males and females, mean height (cm) was 165.8 and 151.3, weight (kg) was 50.2 and 42.9, BMI (kg/m2) was 16.7 and 18.4, WC (cm) was 76.4 and 71.2, HC (cm) was 82.5 and 81.3, waist-hip ratio was 0.97 and 0.87, SBP (mm Hg) was 128.4 and 126.2, and DBP (mm Hg) was 76.2 and 74.8 respectively. The difference was significant (P< 0.05).

Graph I Assessment of clinical parameters

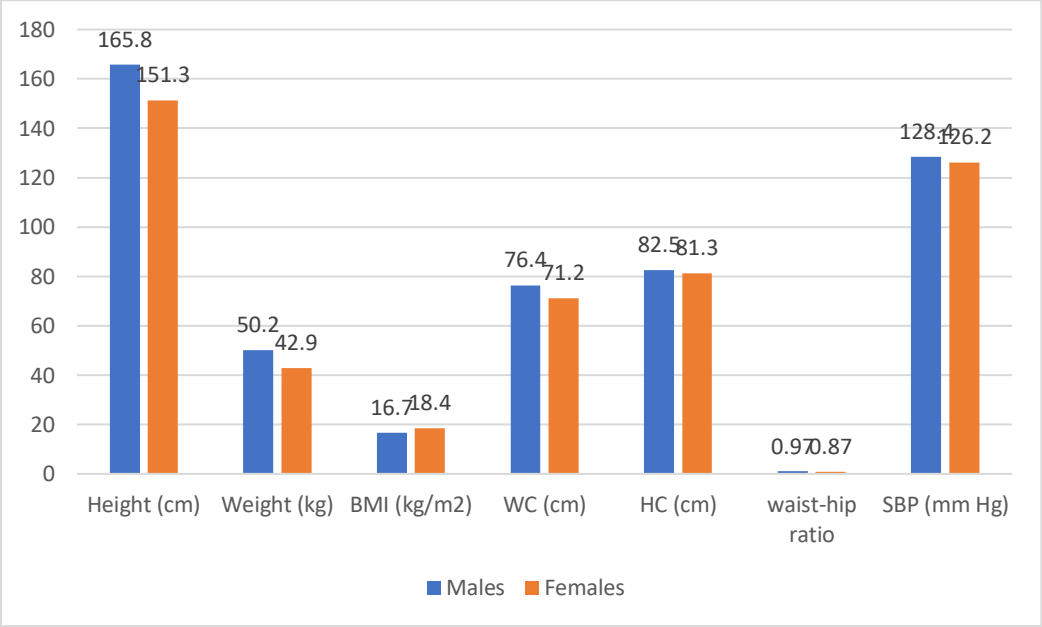

Table III Biochemical parameters

Parameters	Mean	SD
Fasting BG (mg%)	134.8	45.2
Postprandial BG (mg%)	201.4	68.4
HbA1c (%)	7.6	1.2
TC (mg%)	186.2	19.4
TG (mg%)	145.2	25.2
LDL (mg%)	108.4	13.5
HDL (mg%)	49.2	5.7
VLDL (mg%)	32.6	4.2

Table III shows that mean fasting BG (mg%) was 134.8, postprandial BG (mg%) was 201.4, HbA1c (%) was 7.6, TC (mg%) was 186.2, TG (mg%) was 145.2, LDL (mg%) was 108.4, HDL (mg%) was 49.2, and VLDL (mg%) was 32.6.

DISCUSSION

Lean T2DM patients may have dyslipidemia, characterized by elevated triglycerides, decreased HDL cholesterol, and sometimes elevated LDL cholesterol levels.[6]They may also have higher levels of inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6), indicating chronic low-grade inflammation.Some lean T2DM patients may have non-alcoholic fatty liver disease (NAFLD), which is associated with insulin resistance and metabolic dysfunction.[7,8]The present study was conducted to assess clinical and biochemical profiles of lean type 2 diabetes mellitus (T2DM) patients.

We found that out of 84 patients, males were 52 and females were 32. Barma et al[10]evaluated 100 cases of lean type 2 diabetes mellitus (62 males and 38

females). The mean duration of diabetes was 51.7 months (range 5-180 months). The glycemic control was poor according to standard guidelines. The majority of them showed response to oral hypoglycemic agents. Secondary failure to oral hypoglycemic agents was seen in 27 patients. The prevalence of microvascular complications was much higher than macrovascular complications. Neuropathy was the commonest complication seen in 70%, followed by retinopathy in 25%. Only 12 patients had hypertension, one had coronary artery disease and two had cerebrovascular accident. Lipid profile was not significantly deranged in patients.

We found that in males and females, mean height (cm) was 165.8 and 151.3, weight (kg) was 50.2 and 42.9, BMI (kg/m2) was 16.7 and 18.4, WC (cm) was 76.4 and 71.2, HC (cm) was 82.5 and 81.3, waist-hip ratio was 0.97 and 0.87, SBP (mm Hg) was 128.4 and 126.2, and DBP (mm Hg) was 76.2 and 74.8 respectively. Das et al[11] found that 204 patients with NIDDM, 37 were lean and 35 obese. Mean FBG and HbA1C were significantly higher ($P<0.02$ and <0.01) in the former. Serum lipids such as total cholesterol (TC) and triglycerides (TG) were lower ($P<0.05$) in the lean while HDLc values were similar. Eight lean patients and 6 obese (Mean BMI: 15.7 vs.27.4) having similar age (48.0 vs 47.7 years) and mean duration of diabetes (4.6 vs 4.2 years) were subjected to the study of insulin and C-peptide status as well as beta cell reserve. The mean basal serum insulin (IRI) level was lower in the lean (15.3 vs. 28.9 mu u/ml; $P<0.05$) while there was no statistical difference in the basal C-peptide values. Serum samples analysed 2 hours after 75 G of oral glucose and 1 mg I.V. glucagon (Novo) on two consecutive occasions for IRI and C-peptide responses revealed remarkable differences. The rise in IRI was significantly lower ($p<0.01$) in the lean after oral glucose and glucagon as compared to the obese. But the C-peptide values did not reveal significant difference suggesting similar reserve in beta cell function in both these groups of patients with NIDDM.

We found that mean fasting BG (mg%) was 134.8, postprandial BG (mg%) was 201.4, HbA1c (%) was 7.6, TC (mg%) was 186.2, TG (mg%) was 145.2, LDL (mg%) was 108.4, HDL (mg%) was 49.2, and VLDL (mg%) was 32.6. Ikeda et al[12] in their study serum lipid and apolipoprotein levels were assessed in well-controlled and poorly-controlled lean non-insulin-dependent diabetic mellitus (NIDDM) patients without proteinuria or hypertension in order to explore the potential influence of glycaemic management on lipid metabolism. Two groups were created from a sample of 96 lean NIDDM patients (body mass index less than 25 kg m-2 for men and less than 27 kg m-2 for women): group I had a HbA1c concentration of less than 6% for the preceding three months, and group II had a HbA1c concentration of more than 8% for the preceding three months. There were no discernible variations in serum levels of HDL cholesterol, triglycerides, or total cholesterol between groups I and II. There was no discernible difference in the serum levels of apolipoproteins AI, AII, B, CII, CIII, and E between groups I and II. These findings imply that in lean NIDDM patients, glycaemic management had little effect on lipid metabolism.

The limitation of the study is the small sample size.

CONCLUSION

Authors found that lean people with type 2 diabetes mellitus (T2DM) hasdyslipidemia, making the condition a unique clinical entity.

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