

# Comparative Study of Blood Glucose and Glycosylated Hemoglobin Levels and Clinical Profile in Low Body Weight (lean), Nonobese and Obese Type 2 Diabetes Mellitus

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## Research Article

**Abstract: Background:** Diabetes has emerged as a major public health problem in our country. Insulin has important effects on key steps in metabolism of lipids and lipoproteins and altered lipid metabolism is common in diabetic population. As far as diabetes in India is concerned, the vast majority are found to be nonobese. More peculiarly prevalence is rising in lean and underweight persons. Very little data is available on Nonobese type 2 diabetes and especially on those who have low body weight BMI < 18.5 Kg/m<sup>2</sup>. **Materials and Methods:** Present study was carried out in MGM Medical College, Aurangabad. Total 90 known cases of type 2 diabetes mellitus were taken from OPD, Medicine wards of above institute and were divided into three groups as follows: Lean (B.M.I. < 18.5) Nonobese (B.M.I. = 18.5-25) and obese (B.M.I. > 25). B.M.I. (Body mass index): Weight in kg/height in m<sup>2</sup>. Following biochemical parameters were done

1. Fasting and Postprandial Blood glucose measured by glucose oxidase method on RA 1000 fully automated Biochemistry Analyser

2. Glycosylated Hemoglobin was estimated by Flukinger and Winterbalter's colorimetric method.

**Conclusion:** The increase in glycosylated haemoglobin in lean i.e. Group I diabetics as compared to nonobese and obese is statistically significant (P < 0.05). This shows that low body weight type 2 diabetics had a poor glycaemic control as compared to nonobese and obese type 2 diabetics.

**Key Words:** Glycosylated Hemoglobin.

## Introduction

As a discipline diabetes epidemiology is relatively young. The extraordinary high prevalence of type 2 diabetes was reported in Pima Indians in late 1970s and then later in other Pacific and Asian Island populations highlighted the potential for future global epidemic.<sup>7</sup> The predictions were correct and type 2 diabetes has now reached epidemic proportion in many developing nations and disadvantaged

minorities in developed countries. Diabetes has emerged as a major public health problem in our country. Its prevalence rate is increased from 2.1% in 1972 to almost 12% in 1995. Various studies have shown that Indians are having increased propensity to develop diabetes. Impact of industrial and agricultural development in our country with resultant rural to urban migration, improved earning capacity and consequent lifestyle changes may have been the factors responsible for increase in prevalence of diabetes. As far as diabetes in India is concerned, the vast majority are found to be nonobese. More peculiarly prevalence is rising in lean and underweight persons. Very little data is available on Nonobese type 2 diabetes and especially on those who have low body weight BMI < 18.5 Kg/m<sup>2</sup>.<sup>4</sup>

## Materials and Methods

Present study was carried out in MGM Medical College, Aurangabad. Total 90 known cases of type 2 diabetes mellitus were taken from OPD, Medicine wards of above institute and were divided into three groups as follows: Lean (B.M.I. < 18.5) Nonobese (B.M.I. = 18.5-25) and obese (B.M.I. > 25).

B.M.I. (Body mass index) : Weight in kg / height in m<sup>2</sup>

1. Following biochemical parameters were done. Fasting and Postprandial Blood glucose measured by glucose oxidase method on RA 1000 fully automated Biochemistry Analyser.
2. Glycosylated Hemoglobin was estimated by Flukinger and Winterbalter's colorimetric method.

## Observations and Result

**Table 1:** Table showing Summary of Observations and Results and comparison of Blood Glucose and Glycosylated Hemoglobin in Lean, Nonobese and Obese type 2 Diabetes mellitus.

Group		Fasting Blood sugar	Postprandial blood sugar	Glycosylated Haemoglobin
I. Lean Type 2 diabetes	Mean	195.8	283.83	11.98
	S.D.	59.93	8.97	2.62
II. Nonobese type 2 diabetes	Mean	152.1	239.9	8.9
	S.D.	23.87	57.12	1.53
III. Obese Type 2 diabetes	Mean	182.3	279.93	9.93
	S.D.	25.33	34.06	2.02
't Value'		Fasting Blood sugar	Postprandial blood sugar	Glycosylated Haemoglobin
Group I with Group II		7.44	2.82	5.36
Group III with Group II		4.46	3.18	2.15
Group I with Group III		2.25	0.30 *	3.27

(\*P > 0.05, Statistically not significant)

**Table 2:** Table showing Age distribution (n=90)

Group	Number of cases	Age (Yrs) < 30	Age (Yrs) 31-40	Age (Yrs) 41-50	Age (Yrs) 51-60	Age (Yrs) > 60
I	30	1	12	14	2	1
II	30	0	2	11	13	4
III	30	0	2	9	16	3

**Table 3:** Data predicting Weight, Height and Body mass index in Type 2 DM cases (Mean±S.D.)

Group	Weight in Kg	Height in cm	B.M.I. Kg/m <sup>2</sup>
I	40.56± 7.19	139.43± 23.35	17.1± 1.24
II	54.23± 6.30	157.13 ± 9.65	21.92± 1.8
III	64 ± 8.21	152.9 ± 10.74	27.19 ± 1.2

**Table 4:** Table showing Family History

Group	Positive (paternal/maternal)
I	06 (20%)
II	12(40%)
III	15(50%)

**Table 5:** Table showing Socioeconomic status of Type 2 Diabetic patients

Group	Low	Middle	High
I	06 (20 %)	18(60 %)	06(20 %)
II	10 (33.3 %)	15(50 %)	05(16.6 %)
III	07 (23.3%)	13(43.3 %)	10(33.3 %)

**Table 6:** Table showing Duration of Diabetes (years) in Group I, Group II, Group III

Study subjects	Mean	S.D.
Group I	5.7	3.57
Group II	4.8	2.61
Group III	3.9	1.42

**Table 7:** Table showing Urinary Ketones in Group I, Group II, Group III

Study subjects	Ketone bodies
Group I	NIL
Group II	NIL
Group III	NIL

**Table 8:** Table showing Diabetic complications in Group I, Group II, Group III

Complications	Group I	Group II	Group III
Infections	5	3	1
Diabetic neuropathy	5	5	1
Diabetic Cataract	3	4	7

Diabetic Nephropathy	2	1	1
Diabetic Retinopathy	1	2	1
Diabetic Foot	--	--	--
Coronary heart disease	--	1	4
Stroke	1	0	2

**Discussion**

Diabetes mellitus is most prevalent metabolic and noncommunicable disorder in world. In India, a huge number of diabetics are there with BMI less than 18.5. They present with different clinical presentation, morbidity, mortality patterns and biochemical profile when compared to classical patients of NIDDM. 11 There was moderate to severe hyperglycemia in low body weight type 2 diabetics as compared to nonobese and obese type 2 diabetics. The increase in mean fasting blood sugar level in lean was statistically significant (P<0.05) as compared to nonobese and obese type 2 diabetics. The increase in mean postprandial blood sugar level in lean was statistically significant (P<0.05) as compared to nonobese but the increase in mean postprandial blood sugar level in lean was not statistically significant as compared to obese type 2 diabetics. The increase in glycosylated haemoglobin in lean i.e. Group I diabetics as compared to nonobese and is statistically significant(P<0.05). This shows that low body weight type 2 diabetics had a poor glycaemic control as compared to nonobese and obese type 2 diabetics. (Table no. 1) Glycosylated Hb is formed slowly and almost irreversibly by the condensation of two abundant reactants within the red blood cell: glucose and Hb. With continuous accumulation of Glycosylated Hb, the component should be a reflection of average glucose concentration seen by red blood cells during their life span. It has been shown by Keneth H, Gabby et al in 1979 that glycosylation of Hb molecule at various reactive sites increases progressively with increasing hyperglycemia and that chemical measurement of total glycosylation of HbA1 and HbA provides a useful alternative to currently used chromatographic procedures for determination of Glycosylated Hb. 8 Jilal I., Joubert S.M. et al 1982 studied fasting plasma glucose and Glycosylated Hb levels in assessment of diabetic control in NIDDM in young. Response to diet and drug therapy was assessed in a group of 85 Indian patients with NIDDM.HbA1 levels correlated significantly with fasting plasma glucose value. The normal range of Glycosylated hemoglobin is 4 to 7% of total haemoglobin 8. To know the glycaemic control we have measured Glycated haemoglobin in all the three groups. The increase in glycosylated haemoglobin in lean diabetics as compared to nonobese and obese is statistically significant. Das S. et al (1999) carried out study on 380 patients, 91 were found to be having BMI less than 18.5 and were considered as low body weight diabetic patients. They found that these patients were

having poor glycaemic control as the fasting blood glucose was around 200mg% and Glycosylated haemoglobin was more than 10%. 6,7,8 These results match with results of our study .(Table no. 1) Samar Banerjee and Uttam Paul (1999) studied 75 cases of Type 2 Diabetes mellitus. Out of them 25 were lean, 25 nonobese and 25 were obese .They observed poor glycaemic control in lean diabetics as compared to nonobese and obese Type 2 Diabetic patients. Fasting plasma glucose was 238.95±42.26, 197±43.69 and 205.36±40.50 mg% in lean,nonobese and obese diabetics respectively .Postprandial plasma glucose was 294.88±52.28 ,243.81±56.35 and 279.75±52.78 mg%. In lean, nonobese and obese type 2 diabetics respectively. Glycosylated Hb was found to be increased in lean diabetics ie 9.15±0.88 g% as compared to nonobese and obese type 2 diabetics in whom it was 7.65±1.11 and 8.05±1.8% respectively. These results match with results of our study. (Table no.1) Nigam Anant observed that mean fasting and postprandial blood glucose was 209.0±10.6 and 290.0±10.8mg% respectively, in 149 patients with B.M.I.17.2±1.1kg/m<sup>2</sup>. This shows that there is moderately severe to severe hyperglycemia in lean diabetics 9. These results match with results of our study.(Table no.1) Sahay B. K. observed that mean fasting blood sugar levels were 276.0±101.6, 242.63±64.92 and 235.07±91.46 in lean, nonobese and obese type 2 diabetics respectively.10 These observations are similar to observations in our study. 17 (Table no. 1)

**Summary and Conclusions**

Following peculiarities were observed in our study in lean type 2 diabetics:

1. There was moderate to severe hyperglycemia in low body weight type 2 diabetics as compared to nonobese and obese type 2 diabetics. The response to oral hypoglycaemic drugs for longer period showed that they were having good beta cell reserve for insulin. This study ,therefore, demonstrates that these low body weight type 2 diabetics form a definite clinical group and need to be recognized early to improve their metabolic control and thereby quality of life. Establishment of better clinical and laboratory markers such as insulin –C-peptide disparity ie low immunoreactive insulin viz a viz similar C-peptide levels, islet cell antibodies, glucokinase and level of activity of microsomal enzyme systems using drug probes to test fate of insulin in liver, would help in explaining metabolic changes in low body weight type 2 diabetics.12
2. Occurrence of diabetes was found to be a,decade earlier

as compared to nonobese and obese type 2 diabetics.(Table no.2)

3. Incidence more amongst males as compared to females. (Table no.2)

4. Most of them were from middle socioeconomic status and have history of migration from rural to urban area (Table no. 5).

5. Positive family history was found in 20 % of the patients from this group (Table no. 4).

6. In spite of moderate to severe hyperglycemia with poor glycemic control, ketonuria was characteristically absent.(Table no. 7)

7. Infections and neuropathies were common complications. (Table no. 8)

8. In spite of good caloric intake i.e. balanced diet; they were having low body weight with B.M.I. less than 18.5. (Table no.3)

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