Anthropometric and Biochemical Profile of young adults of age group 20-45 years diagnosed newly with type 2 diabetes mellitus at Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi

Harpreet Kour, V. A. Kothiwale1, Shivaprasad S. Goudar

Abstract:

BACKGROUND AND OBJECTIVES: Diabetes mellitus, the most common endocrine disorder is characterized by metabolic abnormalities. India is the second most populated country and presently has the largest share of the adult population in the world. The batteries of papers have reported the increased prevalence of obesity and type 2 diabetes mellitus in the adult population. So it is important to know more about this population, as the early diagnosis will help to develop better strategies for the prevention and management of a disease. Thus the present study has been undertaken to evaluate anthropometric and biochemical parameters in young adults diagnosed newly with type 2 diabetes mellitus.

METHODS: A total of 148 patients of age group 20-45 years with newly diagnosed T2DM were enrolled in the study as per eligibility criteria. The normal healthy controls (n=74) were also enrolled to see the baseline difference. Socio-demographic, Anthropometric and Biochemical parameters were evaluated.

RESULTS: The study population consisted of adult subjects aged 20-45 years newly diagnosed with T2DM. In the present study, most of the diabetics belonged to the overweight and obese category with impaired biochemical parameters.

Keywords: Type 2 Diabetes Mellitus, Obesity, Young adults, Lipid Profile

Diabetes mellitus is considered as one among five primary causes of death in the majority of developed countries, and accumulated evidence shows the increased epidemic proportions in developing countries.[1,2] In India, diabetes has gained the status of a potential epidemic with more than 65.1 million newly diagnosed diabetics. The country is currently facing a very indecisive future about the impending burden imposed by diabetes mellitus.[3,4]

India has also earned the distinguished title as "diabetic capital of the world" with the maximum cases diagnosed with the disease and has now become a global challenge.[2,3] Over the last three decades, there is a change in the status of diabetes, from being considered as a mild disease of the aged individuals to one of the main causes of morbidity and mortality which is affecting the young population. The higher proportion of diabetes in young is observed in the Asian population as compared to the...
western population.[5,14] Recent studies have reported that early onset of diabetes is more progressive from cardiac standpoint that late onset of type 2 diabetes mellitus (T2DM). This may decrease the life expectancy of the patient by almost 15 years.[5–9] Batteries of papers have documented that India is undergoing a speedy epidemiological metamorphosis due to a sedentary lifestyle, advancing age, increased consumption of junk food and diet rich in fats and salts, no exercise regimen, and increased work and emotional stress levels. All these are going in the path of an increased number of patients with obesity and various noncommunicable diseases.[4,10]

Various papers have also documented that obesity in the adult population makes the 2–3 times more vulnerable for lifestyle disorders.[11,12] India is the second most populated country and at present has the largest share of the adult population in the world. It has been documented that India will continue to hold this share for the next 20 years. There is the least data available on young diabetics, so it is important to know more about this population. The detection of characteristics for early onset of diabetes in patients will help toward further development of better strategies and management of this disease. Thus, the present study was undertaken to evaluate anthropometric and biochemical parameters in young adults diagnosed newly with T2DM.

**Objectives of the study**

a. To study anthropometric parameters in young adults of age group 20–45 years diagnosed newly with T2DM on dietary control and oral antidiabetic drugs

b. To study biochemical parameters in young adults of age group 20–45 years diagnosed newly with T2DM on dietary control and oral antidiabetic drugs.

**Materials and Methods**

This study was conducted in the Research Laboratory, Department of Physiology, Jawaharlal Nehru Medical College, KLE Academy of Higher Education and Research, Belagavi. All patients diagnosed newly with T2DM, from Medicine outpatient department of Dr. Prabhakar Kore Hospital and Research Centre, were enrolled in the study after screening for eligibility as per protocol from the period of April 2017 and ended on October 2019. Ethical clearance and written informed consent were obtained before the conduct of the study.

**Inclusion criteria**

All the patients diagnosed newly with T2DM of the age group 20–45 years, treated with only diet and oral antidiabetics.

**Exclusion criteria**

Individual with a history of diabetes more than a year, smokers, and alcoholics.

The sample size was calculated using belownned formula:

\[
\frac{(Z_{1-\beta} + Z_{1-\alpha})^2 (SD_1^2 + SD_2^2)}{(X_1 - X_2)^2}
\]

Where, \( Z_{1-\alpha} \) = at 95%, confidence interval = 1.96, \( Z_{1-\beta} \) = at 80%, power of the test = 1.64, mean and standard deviation (SD) for study and control groups were taken: 29.3 ± 0.84 and 28.7 ± 1.69, \( X_1 - X_2 \) = Expected impact size and \( n = (1.64 + 1.96)^2 (0.8426152^2 + 1.6970562^2) / (29.3–28.7)^2 \approx 130. \) Accounting dropout cases as 10%, then the calculated sample size was \( = 132 / 0.9 = 144.4 \) – rounded to 146.

A total of 74 age-, gender-, and education-matched healthy normal controls were also enrolled to see the baseline difference among study parameters.

Hence, there were two groups in this study. Group I: Normal healthy controls (n = 74) and Group II: Diabetic controls (n = 148).

**Study parameters**

I. Descriptive data of study participants such as age, medical history, duration of diabetes, and any associated health problem were obtained by interviewing the participants

II. Demographic data: Age in years was noted to the nearest completed year as determined from their ration card/driving license/adhar card. Socioeconomic status was evaluated by taking education, occupation, and family income into account and scoring them as per Kuppuswamy’s scorecard[13]

III. Anthropometry Parameters: It included

a. Height (Ht in cm): height in centimeters was measured (to the nearest 0.1 cm) with steel, anthropometric rod, with the individual, standing barefooted in erect position

b. Weight (Wt in kg): weight in kilograms (to the nearest 0.5 kg) was recorded with the individual standing on the weighing scale, barefooted wearing minimum clothes

c. Body mass index (BMI kg/m²): was calculated by Quelet index. The categorization of the BMI was done according to the BMI criteria for the Indian population[14]

d. Body fat percentage was calculated using Siri’s equation

e. Skinfold thickness (in mm) was obtained from seven sites which were measured by Harpenden...
skinfold calipers (Anand agencies, Pune). The mean of two measurements was considered
f. Waist circumference (WC in cm): it was measured midway between iliac crest and lowermost margin of ribs with a nonstretchable measuring tape. According to guidelines, cutoffs for central obesity/abdominal obesity were considered to be present when WC ≥90 cm in males and ≥80 cm in females[15]
g. Hip circumference (HC in cm): measured at the level of the greater trochanters in centimeters
h. Waist–hip ratio (WHR): It was calculated with the corresponding values of WC divided by the hip circumference. The waist–hip ratio of ≥1.0 for males and ≥0.85 for females was considered as truncal obesity.[14]

IV. Biochemical parameters: venous blood was drawn for biochemical examination which included hemoglobin percentage (Hb%), glucose profile, and lipid profile. For fasting blood sugar (FBS), 2 ml of the venous sample was drawn at 8:30 am, following 12 h overnight fast and for postprandial venous sample was drawn after 2 h of breakfast. All the parameters which were under biochemical investigation were determined in the serum of the individuals using commercially available reagent kits.

a. Hb levels (g/dl) were estimated by an automated method
b. Fasting (fasting blood glucose in mg/dl) and postprandial blood glucose (in mg/dl) levels were estimated by glucose oxidase enzymatic method
c. Glycated hemoglobin (HbA1c %) was estimated with ion exchange resin method. The American Diabetes Association (ADA) proposed HbA1c ≥6.5% for the diagnosis of diabetes and 5.7%–6.4% for the highest risk to progress to diabetes
d. Serum total cholesterol (TC mg/dl) was determined by an enzymatic colorimetric method
e. Triglycerides (TGs mg/dl) were determined by an enzymatic method
f. High-density lipoprotein (HDL mg/dl) was estimated by a precipitant method
g. Low-density lipoprotein (LDL mg/dl) and very-LDL (VLDL mg/dl) were calculated using Friedewald’s formula.

The lipid profile of the individuals was classified based on the Adult Treatment Panel III model of National Cholesterol Educational Program and glycemic control as per the criteria laid in 2018 by ADA.[16]

Statistical analysis
Descriptive analysis was carried out by mean and SD for quantitative variables, frequency, and proportion for categorical variables. For normally distributed quantitative parameters, the mean values were compared between study groups using independent sample t-test. All data were analyzed by SPSS (Statistical Package for social sciences, version 20, SPSS, IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp).

Results
The distribution of diabetics and controls with respect to age and gender was found to be same in both groups. Newly diagnosed patients were falling in the lower middle and upper middle class as per socioeconomic status classification and comparatively lower class was least affected. No statistical difference was seen in newly diagnosed diabetic patients among study groups based on religion (Table 1).

54.7% of newly diagnosed patients were between the age group of 36 and 40 years. About 18.9% were between the ages of 41 and 45 years, 13.5% and 11.5% were between 31 and 35 years and 26–30 years while only 1.4% was between 20 and 25 years. Of the total 148 newly diagnosed T2DM patients of age group 20–45 years (males 55.40%, females 44.59%), majority were from the age group 36–40 years (54.7%) (Graph 1).

Table 2 shows the various anthropometric indices among diabetics and controls. The anthropometric values were apparently high in diabetic patients than in controls. It was observed that among diabetics, BMI, body fat percentage, WC, and waist–hip ratio were significantly high as compared to the normal controls (P < 0.001). Whereas no significant difference was found in HC among diabetics and normal controls (P = 0.704). Height between the groups was comparable. Height between two groups was comparable.

Table 1: Sociodemographic characteristics of study sample

| Parameter          | NC (n=74), n (%) | Diabetic population (n=148), n (%) | P*       |
|--------------------|------------------|-----------------------------------|----------|
| Age (mean±SD)      |                  |                                   |          |
| Gender             |                  |                                   |          |
| Male               | 47 (63.51)       | 82 (55.40)                        | 0.248    |
| Female             | 27 (36.48)       | 66 (44.59)                        |          |
| Socioeconomic status |                 |                                   |          |
| Upper middle       | 18 (24.32)       | 53 (35.81)                        | 0.265    |
| Low middle         | 34 (45.94)       | 65 (43.91)                        |          |
| Upper lower        | 16 (21.62)       | 22 (14.86)                        |          |
| Lower              | 6 (8.108)        | 8 (5.405)                         |          |
| Religion           |                  |                                   |          |
| Hindu              | 38 (51.35)       | 78 (52.70)                        | 0.770    |
| Muslim             | 32 (43.24)       | 65 (43.91)                        |          |
| Christian          | 4 (5.405)        | 5 (3.378)                         |          |

NC=Normal control, SD=Standard deviation, *P < 0.05
Sixty-six percent of diabetics were obese and 31% were in preobese category as per BMI classification of obesity. The proportions were very high in diabetics as compared to normal controls [Table 3].

98.6% of diabetics in our study group had high body fat percentage whereas 66% were obese and 33% were overweight in normal controls [Table 4].

79% of diabetics had high WC, which is indicative of abdominal obesity [Table 5].

70% of diabetics had high WHR, which is indicative of truncal obesity [Table 6].

Hb% was comparable in both groups. The mean values of FBS, postprandial blood sugar, and HbA1c were significantly higher in diabetic patients in comparison to normal controls ($P < 0.05$) [Table 7].

### Discussion

The study population consisted of adult individuals aged 20–45 years newly diagnosed with T2DM. In the present study, most of the diabetics belonged to the overweight and obese category (BMI > 30 kg/m²). A multicenter and population-based study from China has reported high BMI and impaired lipid profile were found in patients with T2DM diagnosed at young ages.[17] Despres has also found similar results and reported that a particular range of abdominal obesity is a significant risk factor for the development of T2DM.[18] The study by Koh-Banerjee et al. observed changes in BMI values in early adulthood resulting in the development of diabetes at a younger age.[19] The health professionals follow-up study reported an increased risk of T2DM by 3.5% if weight gain is 8–9 kg from age of 21 years and have also reported increased risk by 7.3% for every 1-kg of weight gained.[20]
The table below summarizes the biochemical parameters of study groups.

| Variables | NC (n=74) | Diabetics (n=148) | t | P |
|-----------|-----------|-------------------|---|---|
| Hb (g %)  | 13.41 (1.43) | 13.47 (1.44) | 0.296 | 0.767 |
| FBS (mg/dl) | 89.96 (6.59) | 105.45 (11.23) | 10.94 | <0.001 |
| PPBS (mg/dl) | 123.85 (13.08) | 140.59 (10.21) | 10.45 | <0.001 |
| HbA1c (%) | 4.93 (0.47) | 6.17 (0.37) | 21.05 | <0.001 |
| TC (mg/dl) | 208.93 (25.46) | 219.57 (26.54) | 2.85 | <0.05 |
| TG (mg/dl) | 118.00 (18.53) | 156.78 (14.78) | 16.88 | <0.001 |
| HDL (mg/dl) | 52.27 (7.72) | 43.23 (7.28) | 9.48 | <0.001 |
| LDL (mg/dl) | 133.00 (27.40) | 145.93 (28.84) | 3.20 | <0.01 |
| VLDL (mg/dl) | 23.66 (3.67) | 31.41 (2.91) | 17.09 | <0.001 |

Hb=Hemoglobin, FBS=Fasting blood sugar, PPBS=Postprandial blood sugar test, HbA1c=Hemoglobin A1c, TC=Total cholesterol, TG=Triglycerides, HDL=High-density lipoprotein, LDL=Low-density lipoprotein, VLDL=Very-low density lipoprotein, NC=Normal control.

In our study, the glucose profile of young diabetic patients was comparatively on a higher range as compared to normal healthy controls. It has been documented in various studies that hyperglycemia in prediabetic states also can lead to the development of macro and microvascular complications with activation of different physiological pathways. The pathophysiological mechanism in postprandial high glucose level is may be due to altered insulin response of beta cells of Langerhans of the pancreas during its first phase of secretion which in turn suppresses the production of endogenous insulin. Beta-cells further deteriorate and thus decrease the production of endogenous insulin. In due course of time, when beta cells fail to produce insulin, the postprandial high sugar induces the production of large amounts of reactive oxygen species which in turn induces apoptosis of beta-cells of pancreas. Once the beta-cells fail, postprandial hyperglycemia may induce large amounts of that can cause further damage to cellular components of insulin production.

In the present study, TGs and VLDL were statistically higher whereas HDL levels were decreased in diabetics. TC and LDL were also comparatively high in diabetics as compared to normal controls. This pattern of lipid profile in diabetics is well documented in middle-aged and elderly population and is found the same in our study population also.

Dyslipidemias are considered an early process in the development of T2DM and are associated with insulin resistance. The earlier studies have reported the pattern of dyslipidemia is characterized by a mild increase in TG levels, decreased HDL cholesterol levels and LDL-cholesterol levels. At the adipose tissue level, the altered action of insulin is because of impaired intracellular hydrolysis of TGs with increase release of nonesterified (free) fatty acids (NEFAs) into the bloodstream. It further increases the influx of NEFAs to the hepatocytes and enhances the synthesis of TGs and VLDL, which results in increased plasma levels of both. The increased TG levels also result in reducing HDL along with a reduction in antioxidant and antiinflammatory activities as it is cardioprotective in nature. Various studies have reported the correlation between glucose and lipid profiles. Impaired functioning of carbohydrate metabolism also leads to impairment in functions of lipid metabolism as both are interrelated to each other. Studies have also reported 70% increased the prevalence of T2DM, 70% in young adults of age group 30–39 years over the last 10 years, making the young population the rapid emerging group for obesity and T2DM.

A study conducted by the Joint Asia Diabetes Evaluation study, which recruited 41,029 Asian participants and 10% of them were from China, suggested the BMI, LDL, and TG levels were higher in early-onset diabetes participants. Similar findings were observed in our study. To the best of our knowledge, this is among few studies on young adults diagnosed with T2DM. Further cohort studies involving these patients should be initiated to understand more clearly about the risk factors and various characteristic features in young diabetic patients.

**Conclusion**

The results of the present study suggest that patients with newly diagnosed T2DM have impaired obesity indices including anthropometric and biochemical parameters. Understanding these in detail can help further to develop better strategies for the prevention and management of this disease in young adults.

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**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Ascaso JF, Parido S, Real JT, Lorente RI, Priego A, Carmena R. Diagnosing insulin resistance by simple quantitative methods in subjects with normal glucose metabolism. Diabetes Care 2003;26:3320-5.
2. Katz A, Nambi SS, Mather K, Baron AD, Follmann DA, Sullivan G, *et al.* Quantitative insulin sensitivity check index: A simple, accurate method for assessing insulin sensitivity in humans. J Clin Endocrinol Metab 2000;85:2402-10.
3. Joshi SR, Parikh RM. India – Diabetes capital of the world:
Now heading towards hypertension. J Assoc Physicians India 2007;55:323-4.
4. Kumar A, Goel MK, Jain RB, Khanna P, Chaudhary V. India towards diabetes control: Key issues. Australas Med J 2013;6:524-31.
5. Cowie CC, Rust KF, Ford ES, Eberhardt MS, Byrd-Holt DD, Li C, et al. Full accounting of diabetes and pre-diabetes in the U.S. Population in 1988-1994 and 2005-2006. Diabetes Care 2009;32:287-94.
6. Xu Y, Wang L, He J, Bi Y, Li M, Wang T, et al. Prevalence and control of diabetes in Chinese adults. JAMA 2013;310:948-59.
7. Constantino MI, Molyneaux L, Limacher-Gisler F, Al-Saeed A, Luo C, Wu T, et al. Long-term complications and mortality in young-onset diabetes: Type 2 diabetes is more hazardous and lethal than type 1 diabetes. Diabetes Care 2013;36:3863-9.
8. Hillier TA, Pedula KL. Characteristics of an adult population with newly diagnosed type 2 diabetes: The relation of obesity and age of onset. Diabetes Care 2001;24:1522-7.
9. Rhodes ET, Prosser LA, Hoerger TJ, Lieu T, Ludwig DS, Laffel LM, et al. Estimated morbidity and mortality in adolescents and young adults diagnosed with type 2 diabetes mellitus. Diabet Med 2012;29:453-63.
10. Prasad AN. Type 2 diabetes mellitus in young need for early screening. Indian Pediatr 2011;48:683-8.
11. IDF Diabetes Atlas, 4th ed. Brussels, Belgium: International Diabetes Federation; 2009. Available from: http: //www. diabetesatlas.org. [Last assessed on 2016 Jan 07].
12. Chan JC, Malik V, Jia W, Kadowaki T, Vijayaraghavan K. Treatment of dyslipidemia in patients with type 2 diabetes. Lipids Health Dis 2010;9:144.
13. Ravi Kumar BP. Kuppuswamy’s socio-economic status scale – A revision of economic parameter for 2012. Int J Res Develop Health 2013;1:2-4.
14. Snehalata C, Viswanathan V, Ramachandra A. A cut off values for normal anthropometric variables in Asian Indian adults. Diabetes care 2003;26:1380-4.
15. Kamat A, Shuvprakash G, Adhikari P. Body mass index and waist circumference in type 2 diabetes mellitus patient attending diabetic clinic. Ind J Biol Med Res 2011;2:636-38.
16. Lipsy RJ. The national cholesterol education program adult treatment panel III guidelines. J Manag Care Pharm 2003;9:2-5.
17. Zhou X, Zhou X, Ji L, Yang W, Lu J, Weng J, et al. The characteristics of newly diagnosed adult early-onset diabetes: A population-based cross-sectional study. Sci Rep 2017;7:46534.
18. Després JP. Abdominal obesity as important component of insulin-resistance syndrome. Nutrition 1993;9:452-9.
19. Koh-Banerjee P, Wang Y, Hu FB, Spiegelman D, Willett WC, Rimm EB. Changes in body weight and body fat distribution as risk factors for clinical diabetes in US men. Am J Epidemiol 2004;159:1150-9.
20. Schienkiewitz A, Schulze MB, Hoffmann K, Kroke A, Boeing H. Body mass index history and risk of type 2 diabetes: Results from the European prospective investigation into cancer and nutrition (EPIC)-Potsdam study. Am J Clin Nutr 2006;84:427-33.
21. Shoelson SE, Lee J, Goldfine AB. Inflammation and insulin resistance. J Clin Invest 2006;116:1793-801.
22. Taylor SI, Kadowaki T, Kadowaki H, Accili D, Cama A, McKeon C, et al. Mutations in insulin-receptor gene in insulin-resistant patients. Diabetes Care 1990;13:257-79.
23. Ceriello A. Postprandial hyperglycemia and diabetes complications: Is it time to treat? Diabetes 2005;54:1-7.
24. Baron AD. Impaired glucose tolerance as a disease. Am J Cardiol 2001;88:161H-9H.
25. Stumvoll M, Goldstein BJ, van Haeften TW. Type 2 diabetes: Principles of pathogenesis and therapy. Lancet 2005;365:1333-46.
26. Praet SF, van Loon LJ. Exercise therapy in type 2 diabetes. Acta Diabetol 2009;46:263-78.
27. Robertson RP, Harmon J, Tran PO, Tanaka Y, Takahashi H. Glucose toxicity in beta-cells: Type 2 diabetes, good radicals gone bad, and the glutathione connection. Diabetes 2003;52:581-7.
28. Firdous, S. Khan MZ. Comparison of patterns of lipid profile in type-2 diabetics and non diabetics. Ann King Edward Med Coll 2007;3:84-7.
29. Gadi R, Samaha FF. Dyslipidemia in type 2 diabetes mellitus. Curr Diab Rep 2007;7:228-34.
30. Adiels M, Olofsson SO, Taskinen MR, Börén J. Overproduction of very low-density lipoproteins is the hallmark of the dyslipidemia in the metabolic syndrome. Arterioscler Thromb Vasc Biol 2008;28:1225-36.
31. Taskinen MR. Type 2 diabetes as a lipid disorder. Curr Mol Med 2005;5:297-308.
32. Garvey WT, Kwon S, Zheng D, Shaughnessy S, Wallace P, Hutto A, et al. Effects of insulin resistance and type 2 diabetes on lipoprotein subclass particle size and concentration determined by nuclear magnetic resonance. Diabetes 2003;52:453-62.
33. Krentz AJ. Lipoprotein abnormalities and their consequences for patients with type 2 diabetes. Diabetes Obes Metab 2003;5 Suppl 1:S19-27.
34. Vijayaraghavan K. Treatment of dyslipidemia in patients with type 2 diabetes. Lipids Health Dis 2010;9:144.
35. Lipton R, Keenan H, Onyemere KU, Freels S. Incidence and onset features of diabetes in African-American and Latino children in Chicago, 1985-1994. Diabetes Metab Res Rev 2002;18:135-42.
36. Zhang J, Yang Z, Xiao J, Xing X, Lu J, Weng J, et al. Association between family history risk categories and prevalence of diabetes in Chinese population. PLoS One 2015;10:e0117044.
37. Yeung RO, Zhang Y, Luk A, Yang W, Sobrepena L, Yoon KH, et al. Metabolic profiles and treatment gaps in young-onset type 2 diabetes in Asia (the JADE programme): A cross-sectional study of a prospective cohort. Lancet Diabetes Endocrinol 2014;2:935-43.