Educational Administration: Theory and Practice
2024,30(3), 2226 - 2231
ISSN:2148-2403
https://kuey.net/

Research Article

Prevalence of Lean Diabetes in West Bengal

Oly Banerjee[1,2,*], Urmimekhala Das, Tiyesh Paul, Siddhartha Singh, Sandip Mukherjee

[1*]Department of Physiology, Serampore College, 9 William Carey Road, West Bengal-712201
[2]Department of Medical Laboratory Technology, School of Allied Health Sciences, Swami Vivekananda University, Bara Kanthalia, West Bengal 700121.

***Corresponding Author:** Oly Banerjee
*Department of Physiology, Serampore College, 9 William Carey Road, West Bengal-712201

Citation: Oly Banerjee et al. (2024), Prevalence Of Lean Diabetes In West Bengal, *Educational Administration: Theory and Practice*, *30*(3), 2226 - 2231, Doi : 10.53555/kuey.v30i3.3674

ARTICLE INFO

ABSTRACT

The prevalence of diabetes mellitus has increased exponentially over the past decade in India and globally. Much has been published on the characteristics of type 2 diabetes mellitus and its association with the epidemic of obesity. But relatively little is known about the incidence of lean diabetes, progression of disease and fate of the patients with low-normal body mass index. This study was conducted to determine the prevalence of lean diabetes among diabetic individuals of three districts of West Bengal [Kolkata, Hooghly and 24 Pgs (N)].
A cross-sectional survey was carried out in three districts of West Bengal during January 2022 to August 2022, among adults aged 30-69 years. All diagnosed T2DM patients within a period of 5 years were included in the study. Lean diabetes was defined as nonketotic diabetes mellitus with a body mass index (BMI) < 18.5 kg/m^2. Among 66 subjects with diabetes, the proportion of lean diabetes was 14% (9 subjects) the mean age of lean diabetics is 48.50 ± 6.30. Total cholesterol, LDL cholesterol and triglyceride levels were found to be lower in lean diabetics as compared to diabetic individuals with BMI 18.5 - 22.9 or BMI>23.0. Among the lean diabetics, tobacco use is very high (67%) as well as 50% of them are required insulin therapy.
In conclusion, the prevalence of lean diabetes was high (14%) among the selected study population.

Keywords: Lean diabetes, insulin resistance, BMI, type II diabetes

Introduction

Diabetes Mellitus (DM) is a persistent metabolic disorder characterized primarily by enduring hyperglycemia. According to ancient Egyptian manuscripts, it stands as one of the earliest recognized diseases in human history, dating back approximately 3000 years based on archaeological evidence. As per the World Health Organization's official definition, diabetes is identified as a metabolic disorder with various causes, marked by chronic hyperglycemia and disruptions in carbohydrate, fat, and protein metabolism resulting from deficiencies in insulin secretion, insulin action, or both (World Health Organization, 2018). The classification of DM into Type I and Type II occurred in 1936, and in 1988, Type II DM was initially linked to the metabolic syndrome. The global prevalence of DM has witnessed a substantial increase in the past decade (Jayawardena *et al.,* 2012).

Diabetes mellitus (DM) is a diverse condition lacking a precise definition, with clinical features exhibiting significant variation both within and among populations. It poses a considerable public health challenge. According to the International Diabetes Federation (IDF), the projected global diagnosis of type 2 DM (T2DM) is expected to reach 784 million people by 2045, compared to 425 million in 2017 (Sun *et al.,* 2022). The substantial increase in prevalence is primarily linked to the obesity pandemic, accounting for 80-90% of cases (Feeley *et al.,* 2012). Notably, 10-20% of individuals with T2DM are non-obese, excluding latent autoimmune diabetes in adults (LADA) and other diabetes forms (Perry *et al.,* 2012). In certain regions, particularly in Asian countries, the prevalence of the non-obese variant can constitute 60-80% of the total T2DM burden (Perry *et al.,* 2012). Some studies indicate that these non-obese individuals face a higher risk of

cardiovascular events compared to their obese counterparts, underscoring the need for a more comprehensive understanding of the pathophysiological mechanisms involved (Chaudhary *et al.,* 2013).

The simplified explanation for the pathophysiology of Type 2 Diabetes Mellitus (T2DM) has traditionally focused on the interplay between beta-cell dysfunction and insulin resistance. However, the specific defects underlying these processes are intricate and not yet fully comprehended. An earlier study (Davies *et al.,* 2018) connected the pathophysiology of T2DM to its treatment, introducing a new dimension to our understanding. Nonetheless, there remains much to unravel. While insulin resistance overlaying defective insulin secretion has been described in T2DM among obese individuals, understanding the pathophysiology in the non-obese has posed a challenge, as many proposed models have been conducted primarily in obese subjects (Stefan *et al.,* 2017). Some studies indicate that even though these non-obese patients may not meet the technical definition of obesity, they exhibit metabolic obesity, implying that similar pathophysiological mechanisms may apply. While this holds true to some extent, significant differences in pathophysiology between obese and non-obese patients need clarification for more precisely defining the management strategies for these individuals (Stefan *et al.,* 2017).

Lean Diabetes (LD) is defined by a BMI less than 18.5 kg/m2 or, in the case of Ketosis Resistant Diabetes of the Young (KRDY), a BMI less than 18.0 kg/m2. However, in studies from high-income countries, higher cutoff values ranging from 18.0 to 24.9 kg/m2 have been employed (George *et al.,* 2015). Previous research has reported that the proportion of lean diabetic participants varied from 7.5% to 21% (Ahuja *et al.,* 1980). LD is linked to an elevated risk of hypoglycemia and mortality. Patients typically exhibit lifestyle habits such as smoking and alcohol consumption, with cardiovascular comorbidities being less prevalent. Limited information is available about lean diabetes, especially in patients with a low-normal body mass index.

Studies conducted in developing countries indicate that these patients often have a history of childhood malnutrition, a lower socioeconomic status, and an early age of onset without ketosis (Ahuja et al., 1980; Akombi *et al.* 2017). Recent research in the United States has revealed that lean, normal-weight diabetes is not uncommon, particularly among minority populations. These patients are predominantly male and exhibit a higher prevalence of insulin use, suggesting rapid beta cell failure. Additionally, they may experience increased overall, cardiovascular, and non-cardiovascular mortality compared to obese diabetic patients. The potential causal mechanisms of this type of diabetes may involve genetic, acquired, and behavioral factors.

Methodology

A cross-sectional survey was conducted in three districts of West Bengal—Hooghly, 24 Parganas (N), and Kolkata—from January 2022 to August 2022. The study focused on adults aged 30 to 69 years. A total of 66 individuals, representing diverse socio-economic statuses and job profiles, were deemed suitable for analysis. Importantly, all subjects included in the study had been diagnosed with diabetes within the last 5 years.

Data for the questionnaire were gathered both through in-home visits and digital means, utilizing messaging applications online. Consent was obtained manually and digitally in the relevant settings.

Biochemical measurements were obtained in two distinct manners:

1. Individuals who already possessed recent basic health data, having been diligent about monitoring their health conditions. However, they lacked certain parameters necessary for our study, as those were not prescribed by their physicians.
2. Subjects were invited to our college for sample collection, involving an overnight fast of 8 hours before the analysis. Subsequently, the samples were analyzed in our laboratory.

For anthropometric data:

1.The participants' weight was measured using a digital weighing machine, and their height was assessed using a stadiometer. Body Mass Index (BMI) was calculated using Quetelet index (Davies and Lucas, 1989).

Regarding health parameters:

- Fasting plasma glucose (FPG) was utilized to screen for diabetes, following the WHO STEPS survey recommendations.
- Diabetes was defined as FPG ≥ 126 g/dl or being on medication.
- Dyslipidemia was characterized by being on medication or meeting one of the following criteria: Total cholesterol ≥ 200 mg/dl, triglycerides ≥ 180 mg/dl, low HDL (<40 mg/dl for males, <50 mg/dl for females), or LDL cholesterol ≥ 100 mg/dl.

Specifically for lean diabetes:

- Lean diabetes was defined as having diabetes and a BMI of < 18.5 kg/m2.

Considerations

- Body weight was measured in light clothing without shoes, using the SECA 877 scale, rounded to the nearest 0.1 kg. Participants were asked to empty their pockets of any accessories or gadgets before measurement.
- Heights were measured without shoes, employing a portable stadiometer, rounded to the nearest 0.1 cm.
- BMI was calculated as weight (kg) divided by height squared (m2).
- Biochemical analysis included concentrations of total cholesterol, low-density lipoprotein (LDL)-cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides, measured using colorimetric test kits (mmol/L).
- Hypercholesterolemia was defined as total cholesterol ≥200 mg/dl, LDL cholesterol ≥160 mg/dl, HDL cholesterol ≤40 mg/dl, triglycerides ≥150 mg/dl, or medication with lipid-lowering drugs.
- HbA1C <7% (53 mmol/mol) was used to indicate optimal blood glucose control in individuals with type 2 diabetes for the preceding two to three years.

Statistical analysis

Descriptive statistics were done. Lipid profile data were presented as Mean±SD. Student's t test was used to analyze lipid profile data. P-value less than 0.05 was considered statistically significant for all tests.

Results

Demographic variables were presented in Table 1. Age (Mean±SD) of the individuals in BMI<18.5, BMI 18.5-22.9 and BMI>23.0 were 48.5±6.3, 52.6±7.2, 50.4±5.1, respectively. Results further revealed that 55.56% of diabetic individuals of BMI<18.5 were in the age group>50 years. In comparison, more than 50% diabetic individuals of BMI 18.5-22.9 and BMI>23.0 group were in the age group <50 years.

Prevalence of lean diabetes was demonstrated in Figure 1. It was noted that 14% of diabetic study participants showed BMI<18.5 (9 out 66 study participants). Lipid profile data of study participants were presented in Figure 2. Notably, total cholesterol, triglyceride and HDL cholesterol did not show significant changes in between BMI<18.5 and BMI≥18.5. However, LDL cholesterol was found to significantly higher in diabetic subjects having BMI≥18.5.

Table 1: Demographic and physiological variables of study participants

Variables	BMI<18.5 kg/m²	BMI 18.5 -22.9 kg/m²	BMI>23.0 kg/m²
n	9 (14%)	27 (41%)	30 (45%)
Age in years (SD)	48.5 (6.3)	52.6 (7.2)	50.4(5.1)
Age group, n (%)			
30-39	2 (22.22%)	7 (25.93%)	4 (13.33%)
40-49	2 (22.22%)	10 (37.03%)	11 (36.67%)
50-59	3 (33.34%)	4 (14.81%)	6 (20%)
60-69	2 (22.22%)	6 (22.22%)	9 (30%)

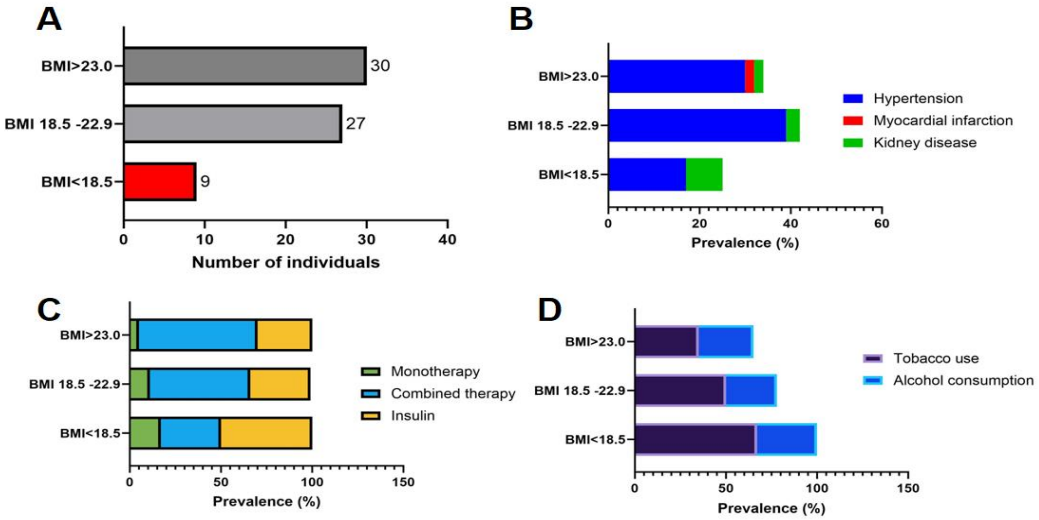

Figure 1: A) Prevalence of lean diabetes, B) Prevalence of hypertension, myocardial infarction and kidney disease C) Use of monotherapy, combined therapy or insulin D) Use of tobacco and alcohol among study participants

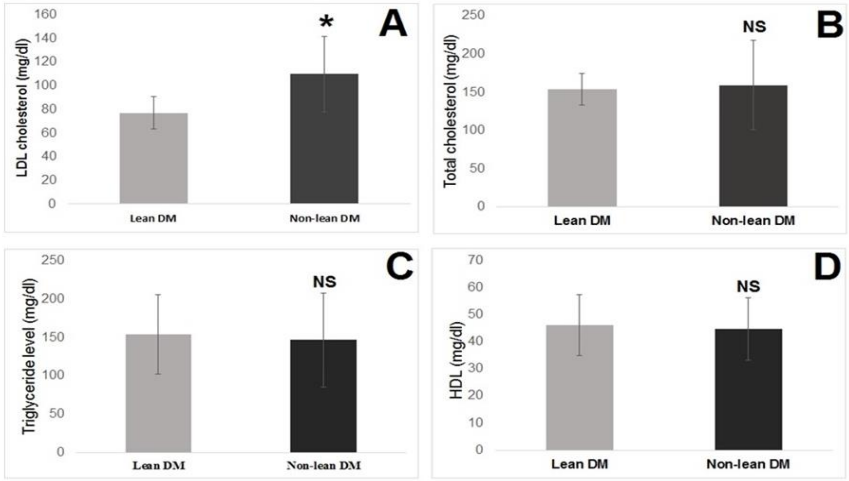

Figure 2: Lipid profile of lean and non-lean diabetic individuals. [A] LDL cholesterol (mg/dl), [B] total cholesterol (mg/dl), [C] triglyceride level (mg/dl), [D] HDL cholesterol (mg/dl). Data presented as mean ± SD. Significance level based on Student's t-test: *p<0.05, NS- Not significant.

Discussion

Occurrence of complications and increased rate of mortality in certain normal weight diabetes patients in comparison to their obese counterparts. This phenomenon is called the "obesity paradox" (George *et al.,* 2015). This "obesity paradox" has also been previously noted in other studies on diabetics and in various other chronic conditions such as hypertension, end stage renal disease and heart failure. It is likely that lower body weight in the presence of obesity related metabolic disorders may just be a reflection of preexisting illness that may predispose to mortality (Carnethon *et al.,* 2012). It is also known that despite having a leaner body mass, cigarette smokers are more insulin resistant, more likely to develop diabetes, and have a higher mortality from chronic lung disease and malignancies as compared with non-smokers (George *et al.,* 2015).
This study aims to document the prevalence of lean diabetes at the population level in the West Bengal, a state in India. The study's strength lies in obtaining estimates of the population with lean diabetes through a community-based approach that identifies individuals diagnosed with diabetes within the last 5 years. The findings reveal that lean diabetes is more prevalent among individuals who tend to be older compared to non-lean diabetic individuals. On the other hand, in the case of obese individuals, they tend to lead a sedentary lifestyle and have occupations that involve moderate to high levels of deskwork (Grams *et al.,* 2015). Certain metabolic characteristics associated with lean diabetes, such as lower levels of hyperlipidemia and a reduced triglycerides/HDL ratio, do not reach statistical significance in this study. This may be attributed to the low number of lean diabetic individuals identified in the cross-sectional survey, comprising only 9 out of 66 individuals, accounting for 14% of the total population.

Contrastingly hypertension was observed to be higher in individuals with lean diabetes compared to their lean counterparts without diabetes. This suggests that, within the lean group, these risk factors were independent of body weight and may be influenced by other factors (Wallace et al., 2004). It was observed earlier that leaner individuals exhibited worse beta cell failure. This leads to the postulation that lean diabetes might represent a distinct variant of Type II diabetes, wherein the failing beta cell struggles to cope even with the modest insulin resistance associated with a lean body weight (Mohan *et al.,* 1997). Similarly, a study conducted across Germany indicated that Lean Diabetes is associated with an elevated risk of hypoglycemia and death (Hartman *et al.,* 2017). These patients are characterized by being male, engaging in lifestyle habits such as smoking and alcohol consumption, while cardiovascular comorbidities are less prominent. These findings are well aligned with the current study as use of tobacco and alcohol found to be higher in diabetic individuals having BMI<18.5.

Compared to patients in other weight groups, those with lean diabetes are more frequently treated with insulin and less frequently with metformin. In type 2 diabetes, beta cells genetically predisposed to fail gradually over the years struggle to cope with increasing insulin resistance associated with obesity. Lean diabetes might be considered a variant of these primary pathogenic mechanisms, characterized by more pronounced and earlier-onset beta cell failure leading to rapid exhaustion. Various potential mechanisms could contribute to beta cell failure, beginning with adverse intrauterine or early postnatal environments that result in a smaller beta cell mass. Genetic predisposition toward a more fragile beta cell mass may lead to early destruction and apoptosis, with studies suggesting that genetic markers of such fragility are more common in lean individuals with diabetes than in their obese counterparts. Higher percentage of insulin users among diabetic individuals of BMI<18.5 group in this study further strengthen these earlier findings.

Furthermore, in developed countries such as the United States, acquired insults like cigarette smoking and alcoholism may represent newly identified and significant contributors to beta cell weakening. In these scenarios, even the minimal insulin resistance associated with a lean body weight could trigger the onset of diabetes. Recent research has also indicated that genetically determined insulin resistance may play a role in the pathogenesis of lean diabetes (Boffetta *et al.,* 2011). Despite the fact that the highest risk of diabetes was observed among the obese individuals, those who were underweight are more likely to have untreated diabetes compared to all other groups. This suggests that lean diabetes is less likely to be detected when compared to individuals who are traditionally considered to be at risk, such as the overweight and the obese.

In conclusion, **t**he present study focused on the prevalence of lean diabetes among individuals diagnosed with diabetes within the last 5 years. One notable finding highlighted in this study is that lean individuals with diabetes required insulin therapy at an earlier stage compared to individuals with normal or obese diabetes.

References

1. Ahuja, M. M. (1980). Diabetes-special problems in developing countries. Bull Deliv Health Care Diabetics Devel Countries, 1, 5-6.
2. Akombi, B. J., Agho, K. E., Hall, J. J., Wali, N., Renzaho, A. M. N., & Merom, D. (2017). Stunting, Wasting and Underweight in Sub-Saharan Africa: A Systematic Review. International journal of environmental research and public health, 14(8), 863. https://doi.org/10.3390/ijerph14080863.
3. Boffetta, P., McLerran, D., Chen, Y., Inoue, M., Sinha, R., He, J., Gupta, P. C., Tsugane, S., Irie, F., Tamakoshi, A., Gao, Y. T., Shu, X. O., Wang, R., Tsuji, I., Kuriyama, S., Matsuo, K., Satoh, H., Chen, C. J., Yuan, J. M., Yoo, K. Y., ... Potter, J. D. (2011). Body mass index and diabetes in Asia: a cross-sectional pooled analysis of 900,000 individuals in the Asia cohort consortium. PloS one, 6(6), e19930. https://doi.org/10.1371/journal.pone.0019930.
4. Carnethon, M. R., De Chavez, P. J., Biggs, M. L., Lewis, C. E., Pankow, J. S., Bertoni, A. G., Golden, S. H., Liu, K., Mukamal, K. J., Campbell-Jenkins, B., & Dyer, A. R. (2012). Association of weight status with mortality in adults with incident diabetes. JAMA, 308(6), 581–590. https://doi.org/10.1001/jama.2012.9282
5. Chaudhary, P., Laloo, D., & Salam, R. (2013). Prevalence of lean type 2 diabetes mellitus in recently diagnosed type 2 diabetes mellitus patients. Indian journal of endocrinology and metabolism, 17(Suppl 1), S316–S317. https://doi.org/10.4103/2230-8210.119645.
6. Davies, J. I., Macnab, A. J., Byass, P., Norris, S. A., Nyirenda, M., Singhal, A., Sobngwi, E., & Daar, A. S. (2018). Developmental origins of health and disease in Africa-influencing early life. The Lancet. Global health, 6(3), e244–e245. https://doi.org/10.1016/S2214-109X(18)30036-6.
7. Davies, P. S., & Lucas, A. (1989). Quetelet's index as a measure of body fatness in young infants. Early human development, 20(2), 135–141. https://doi.org/10.1016/0378-3782(89)90054-6.
8. Feeley, A., Musenge, E., Pettifor, J. M., & Norris, S. A. (2012). Changes in dietary habits and eating practices in adolescents living in urban South Africa: the birth to twenty cohort. Nutrition (Burbank, Los Angeles County, Calif.), 28(7-8), e1–e6. https://doi.org/10.1016/j.nut.2011.11.025.
9. George, A. M., Jacob, A. G., & Fogelfeld, L. (2015). Lean diabetes mellitus: An emerging entity in the era of obesity. World journal of diabetes, 6(4), 613–620. https://doi.org/10.4239/wjd.v6.i4.613.

10. Grams, J., & Garvey, W. T. (2015). Weight Loss and the Prevention and Treatment of Type 2 Diabetes Using Lifestyle Therapy, Pharmacotherapy, and Bariatric Surgery: Mechanisms of Action. Current obesity reports, 4(2), 287–302. https://doi.org/10.1007/s13679-015-0155-x.
11. Hartmann, B., Lanzinger, S., Bramlage, P., Groß, F., Danne, T., Wagner, S., Krakow, D., Zimmermann, A., Malcharzik, C., & Holl, R. W. (2017). Lean diabetes in middle-aged adults: A joint analysis of the German DIVE and DPV registries. PloS one, 12(8), e0183235. https://doi.org/10.1371/journal.pone.0183235.
12. Jayawardena, R., Ranasinghe, P., Byrne, N. M., Soares, M. J., Katulanda, P., & Hills, A. P. (2012). Prevalence and trends of the diabetes epidemic in South Asia: a systematic review and meta-analysis. BMC public health, 12, 380. https://doi.org/10.1186/1471-2458-12-380.
13. Mohan, V., Vijayaprabha, R., Rema, M., Premalatha, G., Poongothai, S., Deepa, R., Bhatia, E., Mackay, I. R., & Zimmet, P. (1997). Clinical profile of lean NIDDM in South India. Diabetes research and clinical practice, 38(2), 101–108. https://doi.org/10.1016/s0168-8227(97)00088-0.
14. Perry, J. R., Voight, B. F., Yengo, L., Amin, N., Dupuis, J., Ganser, M., Grallert, H., Navarro, P., Li, M., Qi, L., Steinthorsdottir, V., Scott, R. A., Almgren, P., Arking, D. E., Aulchenko, Y., Balkau, B., Benediktsson, R., Bergman, R. N., Boerwinkle, E., Bonnycastle, L., … Cauchi, S. (2012). Stratifying type 2 diabetes cases by BMI identifies genetic risk variants in LAMA1 and enrichment for risk variants in lean compared to obese cases. PLoS genetics, 8(5), e1002741. https://doi.org/10.1371/journal.pgen.1002741.
15. Stefan, N., Schick, F., & Häring, H. U. (2017). Causes, Characteristics, and Consequences of Metabolically Unhealthy Normal Weight in Humans. Cell metabolism, 26(2), 292–300. https://doi.org/10.1016/j.cmet.2017.07.008.
16. Sun, H., Saeedi, P., Karuranga, S., Pinkepank, M., Ogurtsova, K., Duncan, B. B., Stein, C., Basit, A., Chan, J. C. N., Mbanya, J. C., Pavkov, M. E., Ramachandaran, A., Wild, S. H., James, S., Herman, W. H., Zhang, P., Bommer, C., Kuo, S., Boyko, E. J., & Magliano, D. J. (2022). IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. Diabetes research and clinical practice, 183, 109119. https://doi.org/10.1016/j.diabres.2021.109119.
17. Wallace, T. M., Levy, J. C., & Matthews, D. R. (2004). Use and abuse of HOMA modeling. Diabetes care, 27(6), 1487–1495. https://doi.org/10.2337/diacare.27.6.1487.
18. World Health Organization. Global report on diabetes. 2016. Available at http://who.int/diabetes/global-report/en/ Accessed 02 Feb, 2018.