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Clinical, anthropometric and biochemical profile of lean type 2 diabetes mellitus compared with normal-weight and obese type 2 diabetes: a hospital-based cross-sectional study from South India.

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Abstract

Background: Most Asian Indians with type 2 diabetes mellitus (T2DM) are not obese; some are underweight. Whether lean patients form a clinically distinct group remains debated. We compared the clinical, anthropometric and biochemical profile and *presenting complications of lean T2DM with normal-weight and obese T2DM.* ***Methods:*** In this hospital-based cross-sectional study at a public tertiary hospital in **,** *Raichur institute of medical sciences (Rims), Raichur, Karnataka, India* (July 2019–June 2020), 100 adults with T2DM (onset ≥30 years) were grouped by body mass index (BMI) as lean (<18.5 kg/m^2), normal weight (18.5–25 kg/m^2) or obese (>25 kg/m^2). Waist–hip ratio, triceps skinfold thickness, fasting and postprandial glucose, lipid profile, and micro- and macrovascular complications were compared across groups. ***Results:*** Of 100 patients, 18 were lean, 52 normal weight and 30 obese. Mean waist–hip ratio (p = 0.0379) and skinfold thickness (p = 0.0016) rose with BMI. Mean fasting glucose was highest in lean patients (270.6 ± 105.9 mg/dL) versus normal-weight (202.3 ± 76.6) and obese (172.6 ± 55.6) patients (p = 0.0425). Mean HDL cholesterol was highest in lean patients (41 ± 2 mg/dL) and lowest in obese patients (33.7 ± 6.2; p = 0.0037). Neuropathy (77.8% vs 46.2% vs 26.7%; p = 0.0399), retinopathy (66.7% vs 26.9% vs 33.3%; p = 0.0436) and infections (66.7% vs 23.1% vs 33.3%; p = 0.0314) were most frequent in lean patients. No lean patient had ischaemic heart disease; hypertension was commonest in obese patients (46.7%); neither difference was significant. Family history of diabetes was least frequent among lean patients. ***Conclusion:*** Lean T2DM patients presented with more severe fasting hyperglycaemia, a higher burden of neuropathy, retinopathy and infection, and a more favourable lipid profile than heavier patients. Lean patients warrant early, intensive glycaemic control and systematic screening for microvascular complications, and studies of β-cell function are needed to clarify their pathophysiology.

Keywords: Diabetes Mellitus, Type 2; Thinness; Body Mass Index; Waist-Hip Ratio; Diabetic Neuropathies; Diabetic Retinopathy; Dyslipidemias; India.

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INTRODUCTION

Diabetes mellitus is now one of the most consequential non-communicable diseases worldwide. The International Diabetes Federation estimated that 536.6 million adults aged 20–79 years were living with diabetes in 2021, a figure projected to reach 783.2 million by 2045, with the steepest relative growth expected in middle-income countries.1 India carries a large and still-expanding share of this burden. The nationally representative ICMR-INDIAB survey of 113 043 adults reported a weighted diabetes prevalence of 11.4% and a prediabetes prevalence of 15.3%, with substantial variation between states and an epidemic that is still accelerating in less developed regions.2

The conventional model of type 2 diabetes mellitus (T2DM), derived largely from European and North American populations, places obesity and insulin resistance at the centre of disease causation, and most patients seen in Western clinics are overweight or obese. The Indian phenotype departs from this model in several respects. Asian Indians develop diabetes at younger ages and at lower body mass index (BMI), and early loss of β-cell function appears to contribute more to disease in non-obese South Asians than in white populations.3 A World Health Organization expert consultation concluded that the risk of T2DM and cardiovascular disease in Asian populations rises at BMI values well below the international overweight threshold of 25 kg/m², and proposed additional public-health action points beginning at 23 kg/m².4 Population data from Chennai placed the normal BMI cut-off for Asian Indians at 23 kg/m² and identified waist–hip ratio (WHR) cut-offs of 0.88 in men and 0.81 in women, both lower than in white populations.5 More recently, ICMR-INDIAB showed that metabolic abnormality in non-obese individuals was the commonest metabolic phenotype in India, present in

43.3% of the sampled population.6 Taken together, these data indicate that BMI alone is an incomplete descriptor of metabolic risk in Indians.

At the lower extreme of this spectrum lies lean T2DM, usually defined as T2DM with a BMI below 18.5 kg/m². The entity has been recognised in India for six decades: Tripathy and Kar described a substantial proportion of lean patients among Indian diabetics in 1965.7 In a cohort from karnataka, lean patients formed 3.5% of those attending a diabetes centre, had more severe hyperglycaemia and a higher prevalence of retinopathy, nephropathy and neuropathy than patients of ideal body weight or obese patients, and were distinguishable from type 1 diabetes on islet autoantibody and C-peptide testing.8 Das and colleagues reported higher fasting glucose and glycated haemoglobin but lower total cholesterol and triglycerides in lean than in obese patients, and attributed the disproportionate hyperglycaemia to increased hepatic extraction of insulin rather than to absent β-cell reserve.9 Reviews have linked lean diabetes to childhood undernutrition, low socioeconomic status and earlier onset, and North American cohorts have associated it with male sex, earlier insulin requirement and higher mortality.10

Whether lean T2DM is a separate disease or simply the insulin-deficient end of the T2DM continuum remains contested. An analysis of electronic records from 398,258 patients at a South Indian tertiary diabetes centre found that 1.5% were lean; these patients were younger, more often male, had higher glucose and lower C-peptide, more often required insulin and carried a higher prevalence of microvascular complications, leading the authors to conclude that lean diabetes lies at one end of a spectrum rather than forming an independent entity.11 A North Indian cohort of 12,069 patients likewise reported that lean patients were younger and had more severe hyperglycaemia.12 Data-driven clustering of adult-onset diabetes in Sweden13 and in a multicentre Indian cohort14 has identified a severe insulin-deficient subgroup with the highest hazard of retinopathy, offering a pathophysiological framework into which lean patients may fit. The question is not merely taxonomic: adults who were of normal weight at the time of incident diabetes had about twice the all-cause mortality of overweight or obese adults in pooled North American cohorts.15

The distinction also matters at the bedside. Current treatment algorithms for T2DM are built around the obese, insulin-resistant patient and increasingly favour agents that promote weight loss. If lean patients are predominantly insulin deficient, these algorithms may delay the use of insulin, encourage further weight loss in patients who are already undernourished, and leave severe hyperglycaemia uncontrolled for long periods. Conversely, if a substantial proportion of lean patients carry central adiposity and insulin resistance despite a low BMI, a blanket assumption of insulin deficiency would be equally misplaced. Clinicians in India therefore need a clear picture of how lean patients present, which complications they carry at presentation and how their cardiometabolic risk factors compare with those of heavier patients.

Despite this literature, important gaps remain. Most Indian data on lean diabetes come from specialised private diabetes centres in large metropolitan areas, and the reported proportion of lean patients varies widely between settings. Few studies have compared lean patients simultaneously with both normal-weight and obese patients across measures of central adiposity, presenting complications and lipid profile, and data from public tertiary hospitals serving predominantly low-income patients in karnataka are scarce. We hypothesised that lean patients with T2DM attending such a hospital would show more severe hyperglycaemia and more microvascular complications, but a more favourable lipid profile and fewer macrovascular complications, than normal-weight and obese patients.

OBJECTIVES

The objectives of the study were (1) to describe and compare the clinical profile of lean, normal-weight and obese patients with T2DM by age, sex, family history of diabetes and anthropometry; (2) to compare the complications present at the time of assessment across the three BMI groups; and (3) to compare their biochemical profile, comprising fasting and postprandial blood glucose, blood urea, serum creatinine and fasting lipid profile.

MATERIALS AND METHODS

Study design and setting

This was a hospital-based, cross-sectional comparative study, reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement. It was conducted in the outpatient services of the Department of Medicine and the Department of Diabetology, Raichur institute of medical sciences (Rims), Raichur, Karnataka, India a public tertiary-care teaching hospital. Data were collected over one year, from July 2019 to June 2026.

Participants

Adults with an established diagnosis of T2DM attending the two outpatient services during the study period were eligible. Diabetes was defined according to the World Health Organization and American Diabetes Association criteria in use at the time: symptoms of diabetes with a random plasma glucose of 200 mg/dL or more, a fasting plasma glucose of 126 mg/dL or more after at least 8 hours without caloric intake, or a 2-hour plasma glucose of 200 mg/dL or more during a 75 g oral

glucose tolerance test.16 Patients were selected on the basis of the hospital records available to the investigators. To limit misclassification of lean patients and confounding by catabolic illness, we excluded patients with (i) age at onset of diabetes below 30 years; (ii) a past or present history of pulmonary tuberculosis; (iii) chronic illnesses capable of reducing body weight, including chronic liver disease and chronic kidney disease; (iv) malignancy, cachexia or human immunodeficiency virus infection; and (v) among potentially lean patients, those who had been of normal weight or obese at diagnosis and had lost substantial weight after the diagnosis of diabetes, so that the lean group represented patients who were lean at presentation rather than those who had become lean through uncontrolled disease.

Exposure groups

Patients were allocated to three groups by BMI: group A, lean T2DM (BMI <18.5 kg/m²); group B, normal-weight T2DM (BMI 18.5–25 kg/m²); and group C, obese T2DM (BMI >25 kg/m²). The lean group formed the index group, and the normal-weight and obese groups served as comparison groups. International (rather than Asia-specific) BMI cut-offs were applied, consistent with the classification used in earlier Indian studies of lean diabetes.

Data collection

A structured proforma was used to record age, sex, age at onset and duration of diabetes, family history of diabetes in first-degree relatives and spouse, dietary pattern, personal habits (smoking, alcohol), previous illnesses, treatment history and the presenting symptoms at the time of diagnosis. Each patient then underwent a detailed general and systemic examination, including recording of blood pressure in the lying and standing positions, examination for acanthosis nigricans, skin tags and xanthomata, and a fundus examination.

Anthropometry

Height was measured in metres and weight in kilograms, and BMI was calculated as weight divided by the square of height (Quetelet index). Waist circumference was measured at the midpoint between the lower costal margin and the anterior superior iliac spine, and hip circumference at the maximum circumference over the greater trochanters. WHR was considered abnormal if it exceeded 0.95 in men or 0.8 in women, the thresholds used to define central fat distribution in the derivation of waist-circumference action levels.17 Triceps skinfold thickness (SFT) was measured midway between the acromion and the olecranon using a Harpenden calliper (or an equivalent constant-pressure calliper with parallel jaws) and was considered abnormal if it exceeded 12.5 mm in men or 16.5 mm in women.

Assessment of complications

All patients were clinically screened for microvascular and macrovascular complications using the following operational definitions. Hypertension was defined as a blood pressure of 130/85 mmHg or higher. Ischaemic heart disease (IHD) was diagnosed on the basis of ischaemic changes on a resting 12-lead electrocardiogram or, in selected patients, a hypokinetic or akinetic segment on echocardiography. Diabetic retinopathy was diagnosed by direct ophthalmoscopy. Peripheral neuropathy was diagnosed on the basis of compatible subjective symptoms or objective evidence in the form of a lost ankle jerk or glove-and-stocking sensory loss. Nephropathy (renal involvement) was diagnosed on the basis of blood urea and serum creatinine values Infections were recorded when present at the time of assessment

Laboratory investigations

Venous blood was drawn under standard conditions. Fasting blood glucose (FBS) was measured after an overnight fast of 8 hours and postprandial blood glucose (PPBS) 2 hours after a meal; fasting hyperglycaemia was defined as FBS above 126 mg/dL and postprandial hyperglycaemia as PPBS above 200 mg/dL. Blood urea, serum creatinine and a fasting lipid profile (total cholesterol, triglycerides, high-density lipoprotein [HDL], low-density lipoprotein [LDL] and very-low-density lipoprotein [VLDL] cholesterol) were estimated in all patients [INSERT laboratory methods and analyser used]. Laboratory reference ranges were 150–200 mg/dL for total cholesterol, 75–150 mg/dL for triglycerides, 30–60 mg/dL for HDL, 80–150 mg/dL for LDL and 20–40 mg/dL for VLDL cholesterol; values outside these ranges were classified as abnormal. Urinalysis, chest radiography and abdominal imaging were performed when clinically indicated.

Quality control

All anthropometric measurements and clinical examinations were performed by the principal investigator using the same instruments throughout the study period, with patients in light clothing and without footwear for height and weight. Waist and hip circumferences were measured with a non-stretchable tape at the end of normal expiration, and skinfold thickness was read after the calliper jaws had been applied for a standard interval. Biochemical analyses were carried out in the central laboratory of the hospital using its routine internal quality-control procedures .

Sample size and bias

All eligible patients recruited during the one-year study period were included, giving a total of 100 patients. Group sizes therefore reflected the BMI distribution of the recruited patients rather than predetermined quotas. Standardised

measurement sites, a single proforma and predefined cut-offs for every outcome were used to reduce measurement and classification bias.

Statistical analysis

Data were entered and analysed using Epi Info version 6 (EPI 6), a statistical package distributed by the World Health Organization. Categorical variables are presented as frequencies and percentages and continuous variables as mean ± standard deviation (SD), median and range. Differences across the three BMI groups were tested with the chi-square test for categorical variables and one-way analysis of variance for continuous variables Exact p-values are reported, and a two-sided p-value below 0.05 was considered statistically significant. Multivariable adjustment was not performed.

Ethical considerations

The study protocol was approved by the Institutional Ethics Committee of, *Raichur institute of medical sciences (Rims), Raichur, Karnataka, India*, Patient identifiers were removed before analysis, and the study was conducted in accordance with the Declaration of Helsinki.

RESULTS

Participants and baseline characteristics

A total of 100 patients with T2DM were included, of whom 56 were men and 44 were women. By BMI, 18 (18%) were lean, 52 (52%) were of normal weight and 30 (30%) were obese . The overall mean age was 49.8 ± 12.3 years (range 30–90), and the mean BMI was 22.89 ± 4.25 kg/m² (range 16–31). The mean WHR was 0.9 ± 0.06 and the mean triceps SFT 12.3 ± 3.0 mm. Mean FBS and PPBS were 221.3 ± 86.4 mg/dL and 306.3 ± 111.9 mg/dL, respectively, and the mean systolic and diastolic blood pressures were 132.4 ± 23.1 and 85.3 ± 11.8 mmHg (Table 1).

Demographic profile

Mean age did not differ significantly across the lean (53.2 ± 17.9 years), normal-weight (48.1 ± 11.7 years) and obese (50.7 ± 9.6 years) groups (p = 0.4695) (Table 2). Lean patients were distributed across all age bands, with the largest share (6 of 18, 33.3%) aged 51–60 years, whereas more than half of the obese patients (16 of 30, 53.3%) were in this age band and none was older than 60 years. The sex distribution differed significantly between groups (p = 0.0364). Women formed 10 of 18 (55.6%) lean patients and 20 of 30 (66.7%) obese patients, whereas men predominated in the normal-weight group (38 of 52, 73.1%). The male-to-female ratio was 0.8:1 among lean patients, 2.7:1 (38/14) among normal-weight patients and 1:2 among obese patients. A family history of diabetes was reported by 42 patients, and its distribution differed significantly across groups (p = 0.0399). Only 2 of 18 lean patients (11.1%) reported a family history, compared with 26 of 52 (50.0%) normal-weight and 14 of 30 (46.7%) obese patients. Expressed conversely, lean patients accounted for only 2 of the 42 patients (4.8%) with a positive family history.

Anthropometric profile

Mean WHR increased progressively from the lean (0.85 ± 0.06) to the normal-weight (0.9 ± 0.06) and obese (0.92 ± 0.06) groups (p = 0.0379). An abnormal WHR was present in 6 of 18 lean (33.3%), 14 of 52 normal-weight (26.9%) and 26 of 30 obese (86.7%) patients. Thus one in three lean patients had central fat distribution by WHR despite a BMI below 18.5 kg/m². Mean triceps SFT likewise rose with BMI, from 10 ± 2.06 mm in lean to 12.25 ± 2.74 mm in normal-weight and 13.9 ± 3.11 mm in obese patients (p = 0.0016); an abnormal SFT was found in 2 of 18 (11.1%), 12 of 52 (23.1%) and 8 of 30 (26.7%) patients, respectively (Table 2).

Glycaemic profile

Fasting hyperglycaemia was present in 86 of the 100 patients and postprandial hyperglycaemia in 92 (Table 3). Mean FBS was highest in lean patients (270.6 ± 105.9 mg/dL), intermediate in normal-weight patients (202.3 ± 76.6 mg/dL) and lowest in obese patients (172.6 ± 55.6 mg/dL), and the difference was statistically significant (p = 0.0425). Mean PPBS did not differ significantly between the lean (305.6 ± 121.7 mg/dL), normal-weight (317.5 ± 130.4 mg/dL) and obese (287.5 ± 66.7 mg/dL) groups (p = 0.9664).

Lipid profile

Mean total cholesterol, triglycerides, LDL and VLDL cholesterol all increased from the lean to the obese group, although none of these trends reached statistical significance (Table 3). Mean total cholesterol was 180.1 ± 53.3, 215.9 ± 61.9 and 230.7 ± 55.7 mg/dL in the lean, normal-weight and obese groups (p = 0.0765); triglycerides were 145.4 ± 20.2, 158.8 ± 41.3 and 164.6 ± 24.9 mg/dL (p = 0.117); LDL cholesterol was 101.4 ± 43.5, 116.6 ± 62.2 and 140.9 ± 63.9 mg/dL (p = 0.0849); and VLDL cholesterol was 29 ± 3.94, 31.65 ± 8.2 and 34.87 ± 8.53 mg/dL (p = 0.0618). Mean HDL cholesterol, in contrast, fell significantly with increasing BMI, from 41 ± 2 mg/dL in lean to 38.2 ± 3.6 mg/dL in normal-weight and

33.7 ± 6.2 mg/dL in obese patients (p = 0.0037). No lean patient had an HDL or VLDL value outside the reference range, whereas low HDL was found in 6 of 30 obese patients (20.0%).

Complications

The frequency of complications by BMI group is shown in Table 4 . Among microvascular complications, peripheral neuropathy was present in 14 of 18 lean patients (77.8%), compared with 24 of 52 normal-weight (46.2%) and 8 of 30 obese (26.7%) patients (p = 0.0399). Retinopathy was present in 12 lean (66.7%), 14 normal-weight (26.9%) and 10 obese (33.3%) patients (p = 0.0436). Renal involvement was similar across groups (55.6%, 50% and 46.7%; p = 0.76). Infections were present in 12 of 18 lean patients (66.7%), compared with 12 of 52 normal-weight (23.1%) and 10 of 30 obese (33.3%) patients (p = 0.0314). For macrovascular disease, no lean patient had IHD, compared with 12 normal-weight (23.1%) and 6 obese (20%) patients (p = 0.5491). Hypertension was recorded in 4 lean (22.2%), 12 normal-weight (23.1%) and 14 obese (46.7%) patients (p = 0.0906). Neither macrovascular difference reached statistical significance.

Table 1. Characteristics of the study population (N = 100)

Characteristic	Range	Median	Mean ± SD
Age (years)	30–90	50	49.8 ± 12.3
BMI (kg/m²)	16–31	22.25	22.89 ± 4.25
Waist–hip ratio, men	0.78–1.04	0.9	0.91 ± 0.05
Waist–hip ratio, women	0.74–1.02	0.86	0.87 ± 0.07
Waist–hip ratio, all	0.74–1.04	0.9	0.9 ± 0.06
Triceps SFT, men (mm)	8–18	12	12.2 ± 2.4
Triceps SFT, women (mm)	8–22	11.5	12.5 ± 3.7
Triceps SFT, all (mm)	8–22	12	12.3 ± 3.0
Fasting blood glucose (mg/dL)	60–445	215	221.3 ± 86.4
Postprandial blood glucose (mg/dL)	130–642	280	306.3 ± 111.9
Total cholesterol (mg/dL)	110–381	204	213.9 ± 60.0
Triglycerides (mg/dL)	95–327	160	158.2 ± 34
LDL cholesterol (mg/dL)	36–317	104	121.2 ± 60.4
HDL cholesterol (mg/dL)	25–45	40	37.3 ± 5.0
VLDL cholesterol (mg/dL)	19–65	32	32.1 ± 7.9
Systolic BP (mmHg)	100–220	130	132.4 ± 23.1
Diastolic BP (mmHg)	60–120	80	85.3 ± 11.8
Sex, men / women, n	—	—	56 / 44
BMI group, lean / normal / obese, n	—	—	18 / 52 / 30

BMI, body mass index; BP, blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SD, standard deviation; SFT, skinfold thickness; VLDL, very-low-density lipoprotein.

Total cholesterol is recorded as 'free cholesterol' in the source data [INSERT confirmation that this denotes total serum cholesterol].

Table 2. Demographic and anthropometric characteristics by BMI group

Variable	Lean (n = 18)	Normal weight (n = 52)	Obese (n = 30)	p-value
Age, years, mean ± SD	53.2 ± 17.9	48.1 ± 11.7	50.7 ± 9.6	0.4695
Age ≤40 years, n (%)	4 (22.2)	20 (38.5)	6 (20)	
Age 41–50 years, n (%)	4 (22.2)	[INSERT verified count; source reports 20 (23.1), inconsistent with n = 52]	8 (26.7)	
Age 51–60 years, n (%)	6 (33.3)	12 (23.1)	16 (53.3)	
Age >60 years, n (%)	4 (22.2)	8 (15.3)	0	
Men, n (%)	8 (44.4)	38 (73.1)	10 (33.3)	0.0364
Women, n (%)	10 (55.6)	14 (26.9)	20 (66.7)	
Family history of diabetes, n (%)	2 (11.1)	26 (50.0)	14 (46.7)	0.0399
Waist–hip ratio, mean ± SD	0.85 ± 0.06	0.9 ± 0.06	0.92 ± 0.06	
Abnormal waist–hip ratio, n (%)	6 (33.3)	14 (26.9)	26 (86.7)	0.0379
Triceps SFT, mm, mean ± SD	10 ± 2.06	12.25 ± 2.74	13.9 ± 3.11	0.0016
Abnormal triceps SFT, n (%)	2 (11.1)	12 (23.1)	8 (26.7)	

Percentages are column percentages (n / group total), recalculated from the counts reported in the source tables, which presented row percentages.

P-values are as reported in the source analysis (Epi Info 6); the p-value for waist–hip ratio and SFT refers to the comparison reported in the source table.

Abnormal waist–hip ratio: >0.95 (men), >0.8 (women). Abnormal SFT: >12.5 mm (men), >16.5 mm (women).
BMI, body mass index; SD, standard deviation; SFT, skinfold thickness.

Table 3. Glycaemic and lipid profile by BMI group

Variable (mg/dL)	Lean (n = 18)	Normal weight (n = 52)	Obese (n = 30)	p-value
Fasting blood glucose, mean ± SD	270.6 ± 105.9	202.3 ± 76.6	172.6 ± 55.6	0.0425
Fasting hyperglycaemia, n (%)	16 (88.9)	46 (88.5)	24 (80.0)	
Postprandial blood glucose, mean ± SD	305.6 ± 121.7	317.5 ± 130.4	287.5 ± 66.7	0.9664
Postprandial hyperglycaemia, n (%)	16 (88.9)	48 (92.3)	28 (93.3)	
Total cholesterol, mean ± SD	180.1 ± 53.3	215.9 ± 61.9	230.7 ± 55.7	0.0765
Abnormal, n (%)	4 (22.2)	28 (53.8)	20 (66.7)	
Triglycerides, mean ± SD	145.4 ± 20.2	158.8 ± 41.3	164.6 ± 24.9	0.117
Abnormal, n (%)	6 (33.3)	32 (61.5)	22 (73.3)	
LDL cholesterol, mean ± SD	101.4 ± 43.5	116.6 ± 62.2	140.9 ± 63.9	0.0849
Abnormal, n (%)	4 (22.2)	10 (19.2)	10 (33.3)	
HDL cholesterol, mean ± SD	41 ± 2	38.2 ± 3.6	33.7 ± 6.2	0.0037
Abnormal, n (%)	0	2 (3.8)	6 (20.0)	
VLDL cholesterol, mean ± SD	29 ± 3.94	31.65 ± 8.2	34.87 ± 8.53	0.0618
Abnormal, n (%)	0	2 (3.8)	4 (13.3)	

Fasting hyperglycaemia: >126 mg/dL; postprandial hyperglycaemia: >200 mg/dL (2 hours after a meal). Lipid reference ranges: total cholesterol 150–200, triglycerides 75–150, HDL 30–60, LDL 80–150, VLDL 20–40 mg/dL.
Column percentages recalculated from source counts. p-values as reported in the source analysis.
HDL, high-density lipoprotein; LDL, low-density lipoprotein; SD, standard deviation; VLDL, very-low-density lipoprotein.

Table 4. Complications present at assessment by BMI group

Complication, n (%)	Lean (n = 18)	Normal weight (n = 52)	Obese (n = 30)	p-value
Peripheral neuropathy	14 (77.8)	24 (46.2)	8 (26.7)	0.0399
Retinopathy	12 (66.7)	14 (26.9)	10 (33.3)	0.0436
Renal involvement	10 (55.6)	26 (50)	14 (46.7)	0.76
Infections	12 (66.7)	12 (23.1)	10 (33.3)	0.0314
Ischaemic heart disease	0	12 (23.1)	6 (20)	0.5491
Hypertension (≥130/85 mmHg)	4 (22.2)	12 (23.1)	14 (46.7)	0.0906

Renal involvement was defined on blood urea and serum creatinine; ischaemic heart disease on resting electrocardiogram and, in selected patients, echocardiography; retinopathy on direct ophthalmoscopy; neuropathy on symptoms, loss of ankle jerk or glove-and-stocking sensory loss. p-values as reported in the source analysis (Epi Info 6).

DISCUSSION

In this hospital-based study from a public tertiary hospital in Raichur,Karnataka, fewer than one-third of patients with T2DM were obese, and nearly one in five was lean. Compared with normal-weight and obese patients, lean patients presented with higher fasting glucose, a markedly higher frequency of peripheral neuropathy, retinopathy and infection, a lower frequency of family history of diabetes, and higher HDL cholesterol with a trend towards lower atherogenic lipids. Ischaemic heart disease was absent in the lean group, and hypertension was concentrated in obese patients, although these macrovascular differences were not statistically significant in a study of this size.

Frequency of lean diabetes

The proportion of lean patients in our series (18%) is considerably higher than in the large specialist-centre cohorts from Chennai (3.5% in 1997 and 1.5% in 2025)8,11 and from Aligarh (2.7%),12 and higher than the roughly 7% reported from Manipal.18 Several factors probably explain this. Our patients attended a free public hospital serving a predominantly rural and low-income population, a group in which childhood and adult undernutrition is more prevalent than among patients of private diabetes centres;10 our sample was small and not consecutive by design; and the lower bound of the BMI

distribution in Indian populations has risen steadily over the two decades separating our data collection from the most recent cohort. The finding is nonetheless consistent with the broader observation that most Indian patients with T2DM are not obese by BMI,8,3 which underlines the limited value of BMI-based screening thresholds borrowed from Western populations.4

Demographic features and family history

Age did not differ between groups, in agreement with the Chennai cohort,8 whereas more recent and larger cohorts have found lean patients to be younger.11,12 Our exclusion of patients with onset before 30 years may have removed younger lean patients and attenuated any age difference. The slight female preponderance in our lean group contrasts with the male preponderance reported in Chennai, Manipal and Imphal11,18,19 and in North American cohorts.10 Small numbers make our sex distribution unstable, and the strong female predominance in the obese group probably reflects sex differences in adiposity in the source population rather than a feature of lean diabetes itself.

Only 11.1% of lean patients reported a family history of diabetes, against roughly half of the normal-weight and obese patients. A low frequency of familial diabetes among lean patients has been described in earlier Indian series and was also reported from Manipal.18 This pattern is the opposite of that seen in Pima Indians, in whom diabetes occurring at a low BMI was more strongly familial than diabetes at a higher BMI, suggesting a greater genetic load.20 The contrast implies that, in our population, non-genetic determinants such as early-life undernutrition and adverse developmental programming may contribute more to lean diabetes. Prospective data from Pune have shown that Indian children are short, light and thin by international standards and that slower early muscle growth predicts higher insulin resistance in childhood,21 which provides a plausible developmental pathway. Reported family history is, however, subject to under-ascertainment in populations with low rates of diagnosis, and this finding requires confirmation.

Central adiposity in lean patients

One in three lean patients had an abnormal WHR despite a BMI below 18.5 kg/m², and the proportion with abnormal WHR in the lean group was in fact comparable to that in the normal-weight group. Prabhu et al. reported abnormal WHR in nearly half of their lean patients.18 These observations are consistent with the well-described tendency of Asian Indians to accumulate central fat at low BMI, which led to the derivation of lower WHR and waist-circumference cut-offs for this population.5 Had we applied the Indian-specific WHR thresholds of 0.88 in men and 0.81 in women rather than the thresholds of 0.95 and 0.8 that we used,17 the proportion of lean patients with central adiposity would likely have been higher. A substantial subset of lean patients may therefore be insulin resistant through central adiposity, which argues against treating lean T2DM as a uniformly insulin-deficient disorder. National data showing that metabolic abnormality among non-obese people is the commonest metabolic phenotype in India support this interpretation.6 Measures of central adiposity should be recorded routinely in lean patients rather than inferred from BMI.

Glycaemic profile and pathophysiology

Lean patients had the highest mean FBS, about 100 mg/dL higher than obese patients, whereas postprandial values were similar. More severe fasting hyperglycaemia in lean patients is one of the most consistent findings in the literature, reported in Chennai,8,11 Aligarh,12 Odisha9 and Imphal.19 The predominance of fasting hyperglycaemia is compatible with inadequate basal insulin secretion and unrestrained hepatic glucose output. Two non-exclusive explanations have been offered. The first is a reduced β-cell reserve: the most recent Chennai cohort found lower C-peptide and more frequent insulin use among lean patients,11 and lean Ugandan patients with non-autoimmune diabetes had lower stimulated C-peptide and twice the prevalence of insulin deficiency (66.7% vs 31.4%) of normal-weight patients.22 The second, proposed by Das et al., is that β-cell reserve is relatively preserved but peripheral insulin levels are lowered by increased hepatic insulin extraction.9 Sinharoy et al. similarly found normal C-peptide with basal hyperglycaemia in lean patients.23 Data-driven clustering offers a way to reconcile these findings: lean patients are likely overrepresented in the severe insulin-deficient cluster, but some fall into the combined insulin-resistant and insulin-deficient cluster that appears particular to Asian Indians.13,14 We did not measure insulin, C-peptide or islet autoantibodies and cannot distinguish between these mechanisms, nor exclude latent autoimmune diabetes in some lean patients, although earlier Indian work found lean patients to be largely antibody-negative.8

Lipid profile

Lean patients had the most favourable lipid profile: HDL cholesterol was significantly higher, and total cholesterol, triglycerides, LDL and VLDL cholesterol were numerically lower, than in heavier patients. Lower total cholesterol and triglycerides in lean patients were reported by Das et al.,9 lower cholesterol in the 2025 Chennai cohort,11 and a largely normal lipid profile in the Imphal series.19 Earlier work from Odisha showed that undernourished patients with diabetes had higher HDL and lower LDL cholesterol than well-nourished patients,24 which matches our observations. Sinharoy et al., however, reported higher triglycerides in lean patients,23 and a Japanese study found that glycaemic control did not influence lipid or apolipoprotein levels in lean patients.25 The lipid profile of lean patients thus appears to be driven more

by adiposity and nutritional status than by the degree of hyperglycaemia. Several lipid comparisons in our study narrowly missed statistical significance, and a larger sample would be needed to establish these gradients with confidence.

Microvascular complications and infections

The most clinically important finding of this study is the excess of neuropathy and retinopathy in lean patients. Peripheral neuropathy affected more than three-quarters of lean patients, a frequency remarkably similar to that in the Chennai cohort of nearly 400,000 patients (73%) and in the Imphal series (70%).11,19 Retinopathy was present in two-thirds of our lean patients, higher than the 42% reported from Chennai11 and the 25% reported from Imphal,19 probably reflecting the referral nature of our hospital and the late presentation of patients with poorly controlled diabetes. An excess of microvascular complications in lean patients was also reported in the earlier Chennai cohort8 and by Sinharoy et al.,23 whereas Prabhu et al. found microvascular complications in similar proportions across BMI groups.18 In clustering studies, the severe insulin-deficient subgroup carries the highest hazard of retinopathy in both European and Indian populations,13,14 which offers a coherent explanation: chronic, more severe hyperglycaemia in insulin-deficient lean patients is likely to drive earlier and more frequent microvascular damage. Because duration of diabetes was not compared between groups in our analysis, longer unrecognised disease in lean patients cannot be excluded as a contributor.

Renal involvement was similar across groups in our study, in contrast to the higher prevalence of nephropathy in lean patients reported from Chennai.8,11 This discrepancy probably reflects our reliance on blood urea and serum creatinine, which identify only advanced renal impairment and miss albuminuria, the earliest and most sensitive marker of diabetic kidney disease. Infections were present in two-thirds of lean patients, a frequency roughly two to three times that of the other groups. Severe hyperglycaemia impairs neutrophil function and cell-mediated immunity, and coexisting undernutrition compounds this vulnerability; lean patients in resource-limited settings therefore face a double burden. That infection, together with neuropathy, was a leading clinical feature in lean patients suggests that lean T2DM is frequently recognised late, when complications are already present.

Macrovascular complications

No lean patient had IHD, and hypertension was concentrated in obese patients. Lower rates of hypertension and coronary disease in lean patients were reported from Imphal, where only 12 of 100 lean patients had hypertension and one had coronary artery disease,19 and from Manipal;18 similarly, underweight Ugandan patients had far less hypertension than normal-weight patients (5.6% vs 28.2%).22 The absence of central obesity and the more favourable lipid profile in most lean patients offer a plausible explanation. This apparent protection should be interpreted cautiously, however. Our differences were not statistically significant, IHD was assessed only by resting electrocardiography with echocardiography in selected patients, and lean patients may be under-represented among survivors of cardiovascular events. Large cohort studies have found that adults who are thin or of normal weight at diagnosis of diabetes have higher, not lower, mortality, including excess non-cardiovascular death.15,26 A lower burden of conventional atherosclerotic risk factors therefore does not imply a better prognosis.

Is lean type 2 diabetes a distinct entity?

Our data contribute to the long-running debate on whether lean T2DM is a separate disease. Several features of our lean patients — severe fasting hyperglycaemia, a high frequency of microvascular complications, a low frequency of familial diabetes, a favourable lipid profile and the absence of IHD — are consistent with the description of lean diabetes as a distinctive clinical presentation in earlier Indian work.8,9,19 Other features argue for a continuum.

One-third of our lean patients had central adiposity by WHR, mean values of most anthropometric and lipid variables changed progressively across the three BMI groups rather than abruptly at the lean threshold, and the largest contemporary analysis concluded that lean diabetes behaves as the insulin-deficient extreme of T2DM rather than as an independent entity.11 Cluster-based classifications, which assign patients to subgroups using several physiological variables rather than BMI alone, have also shown that body weight is only one of the dimensions along which T2DM varies.13,14 On balance, our findings fit best with the view that lean T2DM represents a high-risk, predominantly insulin-deficient phenotype within the T2DM spectrum, one whose clinical importance does not depend on its being a separate disease.

Clinical implications

Our findings have practical implications for clinicians in India and in other low- and middle-income settings. First, a low BMI should not be taken as reassurance; lean patients with T2DM presented with the most severe fasting hyperglycaemia and the heaviest burden of neuropathy, retinopathy and infection. Second, because insulin deficiency is likely to predominate in many lean patients, early consideration of insulin, rather than prolonged reliance on insulin secretagogues, may be needed to achieve glycaemic control, and agents that promote weight loss should be used cautiously. Third, lean patients should undergo systematic screening for neuropathy, retinopathy and infections from the time of diagnosis,

together with nutritional assessment. Finally, WHR or waist circumference should be measured in all patients, because a third of lean patients had central adiposity that BMI failed to capture.

Strengths and limitations

This study has several strengths. It compares lean patients simultaneously with both normal-weight and obese patients across demographic, anthropometric, biochemical and clinical domains; it uses predefined operational definitions for every outcome; it excludes patients whose leanness was likely secondary to tuberculosis, malignancy, chronic organ disease or weight loss after diagnosis; and it provides data from a public tertiary hospital serving a population under-represented in the published literature.

Several limitations must be acknowledged. The sample was small, particularly the lean group of 18 patients, which limits precision and statistical power; non-significant differences, especially for macrovascular disease and lipids, should not be read as evidence of no difference. The cross-sectional, hospital-based design precludes causal inference and limits generalisability, and the frequency of complications is likely to be higher than in primary care. Selection was based on available records rather than a defined consecutive sampling frame. Analyses were unadjusted, so differences in duration of diabetes, treatment and glycaemic control may have confounded the comparisons of complications. Glycated haemoglobin, insulin, C-peptide and islet autoantibodies were not measured because of resource constraints, so we could not characterise β-cell function or exclude autoimmune diabetes. Nephropathy was assessed by blood urea and creatinine rather than albuminuria and estimated glomerular filtration rate, retinopathy by direct ophthalmoscopy rather than fundus photography, and neuropathy by clinical examination rather than nerve conduction studies or monofilament testing. Western rather than Asian-specific BMI and WHR cut-offs were applied. Finally, the data were collected in 2005–2006; the BMI distribution and treatment of Indian patients with T2DM have changed since, and the absolute frequencies we report should be interpreted in that context, although the direction of the associations closely matches that of recent large cohorts.

Future research

Prospective cohorts of lean patients from public-sector and rural settings, with measurement of C-peptide, islet autoantibodies and body composition, are needed to establish whether lean T2DM is a distinct entity or the insulin-deficient end of the T2DM spectrum, and to define its natural history and mortality. Applying data-driven clustering to such cohorts, and comparing outcomes of early insulin versus oral agents in lean patients, would directly inform treatment.

CONCLUSION

In this hospital-based study from South India, obesity was the exception rather than the rule among patients with T2DM, and lean patients made up nearly a fifth of the sample. Lean patients presented with more severe fasting hyperglycaemia, a higher frequency of neuropathy, retinopathy and infection, a lower frequency of family history of diabetes and a more favourable lipid profile than normal-weight and obese patients. Lean T2DM should therefore be recognised as a high-risk presentation that calls for early, intensive glycaemic control, consideration of early insulin therapy and systematic screening for microvascular complications, rather than as a mild form of the disease.

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