RESEARCH PAPER

Clinical Features, Biochemical Profile, and Response to Standard Treatment in Lean, Normal-Weight, and Overweight/Obese Indian Type 2 Diabetes Patients: A Retrospective-Prospective Observational Study

Dr Madhusudhan Rao Chinnamaddiletigari[1], Dr Mrinal Hazarika[2], Dr Meyyammai Chidambaram[3], Dr Anbazhagan Govindraj[4]

[1]Senior Resident, Department of General Medicine, Meenakshi Medical College Hospital & Research Institute, Meenakshi Academy Of Higher Education & Research (Deemed To Be University), Kanchipuram, Tamil Nadu, India
[2]Postgraduate Resident, Department of General Medicine, Meenakshi Medical College Hospital & Research Institute, Meenakshi Academy Of Higher Education & Research (Deemed To Be University), Kanchipuram, Tamil Nadu, India
[3]Assistant Professor, Department of General Medicine, Meenakshi Medical College Hospital & Research Institute, Meenakshi Academy Of Higher Education & Research (Deemed To Be University), Kanchipuram, Tamil Nadu, India
[4]Professor, Department of General Medicine, Meenakshi Medical College Hospital & Research Institute, Meenakshi Academy Of Higher Education & Research (Deemed To Be University), Kanchipuram, Tamil Nadu, India

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) in India presents across all BMI categories. Unlike the predominantly obese Western diabetic phenotype, Indian patients include a sizeable lean and normal-weight subset with distinct clinical and metabolic characteristics. This study compares clinical features, biochemical profiles, and treatment responses across BMI-stratified T2DM groups.

Objectives: To compare clinical features, glycaemic parameters, lipid profiles, complications, and treatment outcomes across lean, normal-weight, and overweight/obese T2DM patients.

Methods: A retrospective-prospective observational study of 90 T2DM patients: Group A – Lean (BMI <18.5, n=20), Group B – Normal weight (BMI 18.5–24.9, n=40), Group C – Overweight/Obese (BMI ≥25, n=30). Study period: 9 months at MMCHRI, Kanchipuram. Data analysed using SPSS 23.0.

Results: Lean patients had higher HbA1c (10.2 ± 1.2%), FBS (270.5 ± 50.8 mg/dL), and PPBS (385.2 ± 52.4 mg/dL), with 85% in poor glycaemic control. Neuropathy (45%) and infections (55%) were highest in lean patients. Insulin requirement was highest in lean patients (60%). Macrovascular complications (hypertension 5%, cardiac 10%, dyslipidaemia 20%) were lowest in the lean group. Obese patients had significantly higher macrovascular burden: hypertension 53.3%, cardiac complications 43.3%, dyslipidaemia 70%.

Conclusion: Lean T2DM in Indians is a distinct entity characterised by severe hyperglycaemia, microvascular and infective complications, and early insulin requirement. Obese T2DM patients carry greater macrovascular risk. BMI-stratified clinical management is essential.

Keywords: Lean diabetes, Type 2 DM, BMI, Indian population, microvascular complications, glycaemic control, insulin, HbA1c

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INTRODUCTION

Diabetes mellitus is a global pandemic affecting an estimated 537 million adults worldwide, with India home to approximately 77 million of these individuals.1 While the classic Western diabetic is obese and insulin-resistant, the Indian diabetic phenotype differs substantially. A significant proportion of Indian patients with T2DM are lean or of normal body weight — a pattern documented widely in the Indian literature and attributed to a combination of genetic predisposition, early-onset insulinopenia, altered hepatic insulin kinetics, and environmental factors.2,3

The prevalence of lean T2DM in India ranges from 1.6% to 26% across published studies.4 Lean T2DM patients are characterised by severe basal hyperglycaemia, relative insulinopenia, a favourable lipid profile with preserved HDL cholesterol, greater susceptibility to microvascular complications (neuropathy, retinopathy) and infections, and relative protection from macrovascular disease (hypertension, ischaemic heart disease).5,6 Despite this, lean and normal-weight T2DM patients remain underrepresented in clinical trials and treatment guidelines.

**Author for Correspondence:* Dr Anbazhagan Govindraj

This study was designed to characterise T2DM patients across BMI categories at MMCHRI, Kanchipuram, comparing their demographic features, glycaemic and biochemical profiles, complication burden, and treatment response, with the aim of generating locally relevant, evidence-based clinical insights.

AIMS AND OBJECTIVES

Primary Objective:

To compare the clinical features, biochemical profile, and response to standard treatment in lean, normal-weight, and overweight/obese T2DM patients.

Secondary Objectives:

1. To compare microvascular (neuropathy, retinopathy, nephropathy) and macrovascular (cardiac, hypertension) complications across BMI groups.
2. To compare glycaemic control (FBS, PPBS, HbA1c) across groups.
3. To assess lipid profile (total cholesterol, HDL, LDL, TGL, VLDL) in relation to BMI.
4. To evaluate the frequency of infective complications and insulin requirement across BMI groups.

MATERIALS AND METHODS

Study Design and Setting

A retrospective-prospective observational study at the Department of General Medicine, MMCHRI, Kanchipuram over 9 months (IEC approval: MMCH & RI IEC/ PG /31/ NOV/ 24).

Sample Size and Grouping

Ninety T2DM patients were classified by WHO BMI criteria: Group A – Lean (BMI <18.5 kg/m², n=20); Group B – Normal weight (BMI 18.5–24.9 kg/m², n=40); Group C – Overweight/Obese (BMI ≥25 kg/m², n=30).

Inclusion Criteria

Age ≥18 years; confirmed T2DM (AHA 2024: HbA1c ≥6.5%, FPG ≥126 mg/dL, 2-h OGTT ≥200 mg/dL, or random plasma glucose ≥200 mg/dL with symptoms); regular antidiabetic medications for ≥6 months.

Exclusion Criteria

T2DM onset <18 years; Type 1 DM, gestational DM, fibrocalculous pancreatic DM, drug-induced DM; irregular medications; pregnancy; pulmonary tuberculosis; chronic liver or kidney disease affecting weight; cancer or HIV.

Data Collection

Structured proforma: demographics, duration of DM, family history, treatment history, presenting complaints. Anthropometry: height, weight, waist, hip (BMI and WHR calculated). Detailed clinical examination for complications. Biochemistry: FBS, PPBS, HbA1c, lipid profile (total cholesterol, TGL, HDL, LDL, VLDL), serum creatinine, BUN. ECG, fundoscopy, urine microalbumin in selected cases.

Diagnostic Criteria

Hypertension: BP >140/90 mmHg. Neuropathy: loss of ankle jerk or glove-and-stocking anaesthesia. Retinopathy: fundoscopy findings. Nephropathy: raised serum creatinine / BUN ± microalbuminuria. Cardiac complication: ischaemic ECG changes or regional wall motion abnormality. Dyslipidaemia: total cholesterol ≥200 mg/dL and/or TGL ≥150 mg/dL and/or low HDL.

Statistical Analysis

SPSS version 23.0. Continuous variables: mean ± SD; categorical: counts and percentages. One-way ANOVA for continuous variables; Chi-square test for categorical variables. $p < 0.05$ = statistically significant.

RESULTS AND OBSERVATIONS

Demographic Profile

Mean age was comparable across groups (Group A: 55.4 ± 7.8 years; Group B: 51.2 ± 9.6 years; Group C: 48.7 ± 10.2 years; p = NS). Female predominance was observed in lean (55%) and normal-weight (55%) groups; males predominated in the obese group (56.7%). Positive family history was significantly lower in the lean group (10%) versus normal-weight (35%) and obese (56.7%) groups ($p < 0.01$), consistent with the lesser genetic loading in lean T2DM.3,5 WHR was abnormal in 40% of lean patients despite low BMI, confirming WHR as a more sensitive marker of central adiposity.

Table 1. Demographic and Anthropometric Characteristics

Parameter	Group A – Lean (BMI <18.5) n=20	Group B – Normal (BMI 18.5–24.9) n=40	Group C – OW/Obese (BMI ≥25) n=30	p-value
Age (yrs) Mean ± SD	55.4 ± 7.8	51.2 ± 9.6	48.7 ± 10.2	NS
Sex – Male	9 (45%)	18 (45%)	17 (56.7%)	<0.05
Sex – Female	11 (55%)	22 (55%)	13 (43.3%)	<0.05
BMI (kg/m²) Mean ± SD	16.9 ± 0.9	21.7 ± 1.8	30.8 ± 3.4	<0.001
WHR Mean ± SD	0.85 ± 0.04	0.90 ± 0.05	0.94 ± 0.04	<0.001
Positive Family History	2 (10%)	14 (35%)	17 (56.7%)	<0.01
Duration of DM (yrs) Mean ± SD	8.3 ± 4.6	9.1 ± 5.2	8.8 ± 5.5	NS

NS = Not Significant. Values = Mean ± SD or n (%). ANOVA for continuous variables; Chi-square for categorical.

Glycaemic and Biochemical Profile

Lean T2DM patients had significantly higher HbA1c (10.2 ± 1.2%), FBS (270.5 ± 50.8 mg/dL), and PPBS (385.2 ± 52.4 mg/dL) compared to other groups ($p < 0.001$). This pattern of severe hyperglycaemia with poor metabolic control is attributed to a reduced beta-cell reserve and relative insulinopenia in lean patients.5,6 Sixty percent of lean patients required insulin therapy, significantly more than in other groups ($p < 0.05$).

Lean patients had a favourably lower lipid profile: total cholesterol (175.6 mg/dL), TGL (110.4 mg/dL), and LDL (100.5 mg/dL), with higher HDL (60.3 mg/dL) compared to obese patients ($p < 0.001$ for most parameters). Blood pressure was lowest in lean patients and highest in the obese group, consistent with the macrovascular burden distribution across groups.

Table 2. Glycaemic and Biochemical Parameters

Parameter	Group A – Lean	Group B – Normal	Group C – OW/Obese	p-value
HbA1c (%) Mean ± SD	10.2 ± 1.2	8.6 ± 1.3	8.3 ± 1.2	<0.001
FBS (mg/dL) Mean ± SD	270.5 ± 50.8	195.3 ± 55.2	178.4 ± 53.6	<0.001
PPBS (mg/dL) Mean ± SD	385.2 ± 52.4	318.6 ± 65.8	296.4 ± 58.3	<0.001
Insulin Use n (%)	12 (60%)	10 (25%)	9 (30%)	<0.05
Total Cholesterol (mg/dL)	175.6 ± 22.4	215.8 ± 28.6	238.4 ± 28.2	<0.001
Triglycerides (mg/dL)	110.4 ± 30.2	185.6 ± 55.4	225.8 ± 62.4	<0.001
HDL (mg/dL)	60.3 ± 8.6	52.4 ± 9.8	42.6 ± 8.8	<0.05
LDL (mg/dL)	100.5 ± 22.4	135.6 ± 28.4	165.4 ± 32.6	<0.001
VLDL (mg/dL)	22.1 ± 6.0	37.1 ± 11.1	45.2 ± 12.5	<0.001
S. Creatinine (mg/dL)	1.1 ± 0.3	1.2 ± 0.4	1.3 ± 0.4	NS
BUN (mg/dL)	22.4 ± 5.8	26.8 ± 8.2	30.1 ± 9.6	NS
SBP (mmHg) Mean ± SD	118.4 ± 12.2	135.6 ± 14.8	148.6 ± 16.4	<0.001
DBP (mmHg) Mean ± SD	74.8 ± 7.2	82.6 ± 9.4	90.4 ± 10.6	<0.001

Values = Mean ± SD or n (%). $p < 0.05$ considered statistically significant.

Complication Profile

Neuropathy was highest in the lean group (45% vs 25% vs 16.7%; $p < 0.01$). Infective complications were most common in lean patients (55% vs 30% vs 16.7%; $p < 0.001$). These findings align with Mohan et al.5 and Gohel et al.6 Retinopathy and nephropathy did not differ significantly across groups (p = NS).

Macrovascular complications showed the expected inverse pattern: hypertension (5% vs 45% vs 53.3%; $p < 0.001$), cardiac complications (10% vs 37.5% vs 43.3%; $p < 0.05$), and dyslipidaemia (20% vs 50% vs 70%; $p < 0.001$) were all lowest in lean patients and highest in the obese group, confirming the cardiovascular protection conferred by the favourable lean metabolic profile.

Table 3. Complication Profile by BMI Group

Complication	Group A – Lean n=20	Group B – Normal n=40	Group C – OW/Obese n=30	p-value
Neuropathy	9 (45%)	10 (25%)	5 (16.7%)	<0.01
Retinopathy	7 (35%)	16 (40%)	7 (23.3%)	NS
Renal Complication	5 (25%)	18 (45%)	10 (33.3%)	NS
Cardiac Complication	2 (10%)	15 (37.5%)	13 (43.3%)	<0.05
Infections	11 (55%)	12 (30%)	5 (16.7%)	<0.001
Hypertension	1 (5%)	18 (45%)	16 (53.3%)	<0.001
Dyslipidaemia	4 (20%)	20 (50%)	21 (70%)	<0.001

Values = n (%). p-values by Chi-square test. NS = Not Significant.

Glycaemic Control and Treatment Response

Poor glycaemic control (HbA1c ≥ 9%) was highest in the lean group (85%) compared to 42.5% in normal-weight and 36.7% in obese patients. No lean patient achieved good glycaemic control during the study period, confirming the difficulty of glycaemic target attainment in lean T2DM and the need for early, intensive insulin therapy in this group.

Table 4. Glycaemic Control Status by BMI Group

Glycaemic Control	Group A – Lean n=20 (%)	Group B – Normal n=40 (%)	Group C – OW/Obese n=30 (%)
Good (HbA1c <7.5%)	0 (0%)	5 (12.5%)	4 (13.3%)
Fair (HbA1c 7.5–8.9%)	3 (15%)	18 (45%)	15 (50%)
Poor (HbA1c ≥9%)	17 (85%)	17 (42.5%)	11 (36.7%)

Good = HbA1c <7.5% | Fair = HbA1c 7.5–8.9% | Poor = HbA1c ≥9%.

DISCUSSION

This study of 90 T2DM patients across three BMI groups confirms that lean T2DM in India is a clinically and metabolically distinct entity. The high prevalence of lean T2DM in our cohort (22.2%) is consistent with reported ranges of 1.6–26% in Indian studies.4

Lean T2DM patients demonstrated severe hyperglycaemia (mean HbA1c 10.2%, FBS 270.5 mg/dL) and poor metabolic control despite treatment, with 60% requiring insulin. This insulinopenic character has been attributed by Das et al. to hyperactivity of hepatic futile cycles and enhanced first-pass insulin extraction, resulting in peripherally low insulin levels.5 Early initiation of insulin is therefore a pragmatic and evidence-supported management approach for lean T2DM.

The high rates of neuropathy (45%) and infections (55%) in lean patients are consistent with Mohan et al.5 (reporting similar increases in microvascular complications) and Gohel et al.6 (reporting neuropathy 52%, infections 42% in lean patients). Severe chronic hyperglycaemia drives oxidative stress, impairs immune function, and damages the microvasculature — mechanisms amplified in lean patients by the duration of uncontrolled hyperglycaemia. Conversely, the low rates of hypertension (5%), cardiac complications (10%), and dyslipidaemia (20%) in lean patients support the concept that the metabolic syndrome and atherosclerotic pathway — typically activated by visceral adiposity and dyslipidaemia — are less operative in this group.

The finding that 40% of lean patients had abnormal WHR despite low BMI reinforces WHR as a superior indicator of central adiposity and cardiometabolic risk in this population. This is consistent with published evidence3 and should inform clinical practice: lean patients with abnormal WHR may still carry some visceral adiposity-related risk and warrant monitoring for macrovascular complications.

The markedly better lipid profile in lean patients (lower TGL, higher HDL, lower LDL) compared to obese patients supports the atherogenic protection documented in lean T2DM by Das et al.,5 Japanese data by Ikeda et al.,7 and the parent MGR study by Shavana et al.8 The absence of dyslipidaemia and lower blood pressure burden in lean patients suggests a different therapeutic approach may be needed — one focused on aggressive glycaemic control and microvascular surveillance rather than lipid management and blood pressure optimisation.

This study's limitations include its single-centre retrospective-prospective design (selection bias risk), modest sample size, and the unavailability of HbA1c, insulin assay, C-peptide, and GAD antibody data for all patients — which would allow more definitive mechanistic classification.

CONCLUSION

Lean T2DM in the Indian population is a clinically distinct entity characterised by severe hyperglycaemia, poor glycaemic control, higher rates of neuropathy and infective complications, and a disproportionate need for insulin therapy — despite a relatively protective lipid and blood pressure profile. Overweight and obese T2DM patients carry a greater macrovascular burden including hypertension, dyslipidaemia, and cardiac complications.

These findings underscore the importance of BMI-stratified clinical assessment of Indian T2DM patients. Lean diabetics require early aggressive glycaemic management with timely insulin initiation and proactive screening for microvascular complications. Obese patients require aggressive management of cardiovascular risk factors. Future multicentre prospective studies with comprehensive biochemical profiling (C-peptide, insulin levels, GAD antibodies) are warranted to fully characterise lean T2DM as a distinct pathophysiological entity and to guide targeted therapy.

DECLARATIONS

Ethics Approval: Institutional Ethics Committee, MMCHRI, Kanchipuram (Protocol submitted: 21.10.2024), (IEC approval: MMCH & RI IEC/ PG /31/ NOV/ 24).

Informed Consent: Written consent obtained from all prospective participants.

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Conflict of Interest: The authors declare no conflict of interest.

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ABBREVIATIONS

BMI – Body Mass Index | WHR – Waist-Hip Ratio | T2DM – Type 2 Diabetes Mellitus | FBS – Fasting Blood Sugar | PPBS – Post-Prandial Blood Sugar | HbA1c – Glycated Haemoglobin | HDL – High Density Lipoprotein | LDL – Low Density Lipoprotein | TGL – Triglycerides | VLDL – Very Low Density Lipoprotein | BUN – Blood Urea Nitrogen | SBP – Systolic Blood Pressure | DBP – Diastolic Blood Pressure | OHA – Oral Hypoglycaemic Agent | DM – Diabetes Mellitus | NS – Not Significant | SD – Standard Deviation | SPSS – Statistical Package for Social Sciences | MMCHRI – Meenakshi Medical College Hospital and Research Institute | IEC – Institutional Ethics Committee | AHA – American Heart Association | WHO – World Health Organization | OGTT – Oral Glucose Tolerance Test

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