ANS-LHS

OPEN ACCESS

Advanced Natural Sciences: Life and Health Sciences,
ISSN: 3107-636X, Vol-3, Issue-2,
July-Dec, 2026, 13-32
https://www.indjournals.com

Research Article

Association between Body Mass Index and Glycated Haemoglobin (HbA1c) in Newly Diagnosed Type 2 Diabetes Mellitus: A Cross-Sectional Analysis from a Tertiary Care Centre in Kanpur, India

Anjaly Mary Varghese[1*], Nilam Nigam[1], Kriti Jalota[1] and Shrawan Kumar[2]

[1]Department of Pharmacology, [2]Department of General Medicine,
Rama Medical College Hospital and Research Centre, Mandhana,
Kanpur Nagar, Uttar Pradesh-209217, India.

Running title: BMI class and HbA1c in newly diagnosed type 2 diabetes

Trial registration: Clinical Trials Registry – India, CTRI/2024/10/074542

Article Info:

Correspondence:
Anjaly Mary Varghese,
Department of Pharmacology, Rama Medical College Hospital and Research Centre, Mandhana, Kanpur Nagar, Uttar Pradesh-209217, India.
Email: niya4niky@gmail.com

DOI: 10.5281/zenodo.22080825

[Received 22-06-2026,
Accepted 11-08-2026,
Published 24-08-2026]

Abstract

Background: Obesity is regarded as the dominant modifiable driver of type 2 diabetes mellitus (T2DM), and lifetime-risk estimates from Western cohorts rise steeply across the body mass index (BMI) spectrum. Whether the *severity of hyperglycaemia at the moment of diagnosis* tracks with BMI in an Indian population — a population characterised by thin-fat phenotype, early beta-cell failure and diabetes occurring at lower BMI — is far less certain.

Objectives: To describe the BMI distribution and the glycated haemoglobin (HbA1c) distribution of newly diagnosed T2DM patients attending the out-patient department of a tertiary care teaching hospital in Kanpur, India, and to test whether HbA1c category is associated with BMI class.

Methods: This was a cross-sectional analysis of the baseline demographic and laboratory dataset of 410 newly diagnosed, treatment-naïve adults with T2DM enrolled in a randomised controlled trial (CTRI/2024/10/074542) between October 2024 and September 2025. Height, weight and BMI were recorded using standardised anthropometry, and BMI was classified into five categories. HbA1c was measured on venous blood and grouped into five glycaemic bands (<6.5%, 6.5–7.4%, 7.5–8.9%, 9.0–9.9%, ≥10.0%). Continuous variables are summarised as median and inter-quartile range (IQR), with class-specific medians, IQRs and observed ranges displayed graphically; the association between BMI class and HbA1c category was tested using Pearson's chi-square test of independence, with $p < 0.05$ considered statistically significant.

Results: The cohort comprised 410 newly diagnosed patients. HbA1c at diagnosis was high across the whole cohort (median 8.1%, IQR 7.0–9.3%), and 15.4% of patients presented with HbA1c ≥10.0%. BMI was distributed across all five classes, with a substantial proportion of patients in the normal-weight (33.7%) and underweight (5.9%) categories in addition to overweight (29.8%) and obese (30.7%) categories. HbA1c category showed no statistically significant association with BMI class ($\chi^2 = 14.3028$, degrees of freedom = 16, $p = 0.576164$; Cramér's V = 0.093). Median HbA1c spanned less than one percentage point across the five BMI classes (8.0–8.9%), the inter-quartile ranges overlapped

Cite this article: Anjaly Mary Varghese, Nilam Nigam, Kriti Jalota and Shrawan Kumar, 2026. Association between Body Mass Index and Glycated Haemoglobin (HbA1c) in Newly Diagnosed Type 2 Diabetes Mellitus: A Cross-Sectional Analysis from a Tertiary Care Centre in Kanpur, India. Advanced Natural Sciences: Life and Health Sciences, 3(2), 13-32.

almost completely, and BMI and HbA1c were essentially uncorrelated on their continuous scales (Spearman ρ = −0.002, p = 0.97).

Conclusion: In this North Indian tertiary-care population, newly diagnosed T2DM occurred — and presented with comparable glycaemic severity — across the entire BMI spectrum. BMI alone did not discriminate patients with severe from those with mild hyperglycaemia at diagnosis. These findings support ethnicity-specific screening strategies that do not rely on a BMI threshold as a gate to diabetes testing, and they reinforce the argument that body-fat distribution and beta-cell reserve, rather than BMI, drive the Indian T2DM phenotype.

Keywords: Body mass index; Glycated haemoglobin; HbA1c; Type 2 diabetes mellitus; Newly diagnosed diabetes; Lean diabetes; Asian Indian phenotype; Cross-sectional study; Kanpur.

Introduction

Diabetes is a major and rapidly increasing global public-health problem, with South Asia contributing substantially to projected growth [1]. The ICMR–INDIAB national study reported a weighted diabetes prevalence of approximately 11.4% and a prediabetes prevalence above 15%, with marked inter-state variation [2]. Earlier phase-I data documented substantial urban–rural variation across the first four study regions [3]. Reviews of the Indian epidemic describe earlier onset, high prevalence at lower body mass index (BMI), and a prominent contribution from central adiposity [4], features that are shared across several Asian populations [5].

BMI remains the most widely used clinical and epidemiological surrogate of adiposity, and the conventional categories range from underweight (<18.5 kg/m²) to class III obesity (≥40.0 kg/m²) [6]. In United States lifetime-risk models, diabetes risk increased sharply across BMI strata [7]; prospective cohorts in women [8] and men [9] likewise demonstrated strong gradients with BMI, adult weight gain and central adiposity. Biologically, excess adiposity promotes insulin resistance and beta-cell stress through ectopic lipid deposition, inflammation and altered adipokine signalling [10].

BMI, however, measures mass relative to height rather than fat quantity or distribution. Asian populations have more body fat at a given BMI than European-origin populations [11], and the BMI–body-fat relationship varies substantially by ethnicity and age [12]. A World Health Organization consultation consequently proposed lower action points for Asian populations [13], while the Asian-Indian consensus classifies BMI 23.0–24.9 kg/m² as overweight or at risk and BMI ≥25.0 kg/m² as obesity [14]. These differences make direct application of Western BMI thresholds to Indian populations potentially misleading.

HbA1c reflects average glycaemia over the preceding weeks and provides a clinically useful measure of metabolic severity at diagnosis. Whether BMI predicts presenting HbA1c among newly diagnosed, treatment-naïve Indian adults remains uncertain. Most available Indian reports combine treated and untreated patients, assess adiposity without stratifying HbA1c, or examine diabetes incidence rather than glycaemic severity once diabetes is established. Evidence from North Indian tertiary-care populations is particularly limited.

This study therefore examined the relationship between BMI class and HbA1c in newly diagnosed, treatment-naïve adults attending a tertiary-care teaching hospital in Kanpur. The objectives were to describe the distributions of BMI and HbA1c, compare classification under international and Asian-Indian BMI thresholds, and test whether HbA1c category was associated with BMI class.

Materials and Methods

Study design and reporting framework

This was a hospital-based, observational, analytical cross-sectional study nested within the

baseline phase of an ongoing randomised controlled trial. The presented data of 410 adults with newly diagnosed type 2 diabetes mellitus were analysed from the demographic dataset of the randomised controlled trial registered with the Clinical Trials Registry – India (CTRI registration number: CTRI/2024/10/074542). Because no intervention had been allocated or administered at the time of the measurements analysed here, all data reported in this article represent the untreated, pre-randomisation state of the cohort. The study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies, and the relevant STROBE domains — setting, participants, variables, data sources, bias, study size, quantitative variables and statistical methods — are addressed in the subsections below [15].

Study setting and duration

The study was conducted in the out-patient departments of General Medicine and Pharmacology at Rama Medical College Hospital and Research Centre, Mandhana, Kanpur, Uttar Pradesh — a tertiary care teaching hospital serving an urban, peri-urban and rural catchment in the Kanpur–Bithoor corridor of central Uttar Pradesh. Recruitment and data collection extended over a twelve-month period, from October 2024 to September 2025 inclusive. A full-year recruitment window was chosen deliberately, so that any seasonal variation in dietary pattern, physical activity, festival-related weight change and health-seeking behaviour would be distributed across the cohort rather than concentrated in a single season, since such variation can measurably shift both anthropometry and HbA1c.

The catchment population is served by a mixed public–private health economy in which diabetes is frequently detected opportunistically — during evaluation of unrelated complaints, pre-operative screening, or symptom-driven presentation — rather than through organised population screening. This context is relevant to the interpretation of the HbA1c distribution reported here, because opportunistic detection systematically delays diagnosis and shifts the presenting HbA1c upward.

Participants: eligibility criteria

Inclusion criteria

- Adults aged 18 years or older of either sex.
- Newly diagnosed type 2 diabetes mellitus, diagnosed within the preceding four weeks and confirmed according to current American Diabetes Association diagnostic criteria — fasting plasma glucose ≥126 mg/dL, or two-hour post-load plasma glucose ≥200 mg/dL on a 75 g oral glucose tolerance test, or HbA1c ≥6.5%, or a random plasma glucose ≥200 mg/dL in the presence of classical hyperglycaemic symptoms [16].
- Treatment-naïve status with respect to all glucose-lowering pharmacotherapy, including oral agents, injectable incretin-based agents and insulin.
- Willing and able to provide written informed consent and to attend follow-up for the parent trial.

Exclusion criteria

- Type 1 diabetes mellitus, latent autoimmune diabetes of adults, gestational diabetes, or secondary/monogenic diabetes (pancreatic, endocrine, drug-induced or syndromic).
- Any prior or current glucose-lowering therapy, or prior documented diagnosis of diabetes.
- Pregnancy or lactation.
- Conditions that invalidate HbA1c interpretation, including haemoglobinopathies and thalassaemia trait, iron-deficiency or megaloblastic anaemia with haemoglobin below the laboratory-defined acceptable threshold, haemolytic disorders, recent blood transfusion within three months, active blood loss, erythropoietin therapy, chronic kidney

disease stage 4–5, and advanced chronic liver disease — each of which alters erythrocyte survival or glycation kinetics and can produce spuriously low or high HbA1c values.

- Conditions that invalidate BMI interpretation, including gross oedema, ascites, anasarca, advanced heart failure, pleural effusion, pregnancy, limb amputation, severe kyphoscoliosis, or inability to stand unassisted for height measurement.
- Systemic glucocorticoid, antipsychotic or antiretroviral therapy known to induce hyperglycaemia, and any acute intercurrent illness, sepsis or diabetic emergency at the time of assessment, since acute stress hyperglycaemia distorts baseline characterisation.

Sampling strategy and sample size

Consecutive eligible patients presenting to the out-patient department during the recruitment window were screened, and all who satisfied the criteria and consented were enrolled, yielding a final analytic sample of 410 participants. The sample size was determined by the requirements of the parent randomised controlled trial rather than by an independent power calculation for the present association analysis; the cross-sectional analysis therefore uses a complete-enumeration approach to the available baseline dataset. In the resulting 5 × 5 table, the mean expected count was 16.4; five of 25 cells (20%) had expected counts below five and none had an expected count below one, meeting Cochran's criteria at the conventional boundary. No separate prospective power calculation was performed for this secondary cross-sectional analysis; results are therefore interpreted from the observed effect sizes, p values and concordance of the supportive analyses rather than from a post hoc claim of achieved power.

Anthropometric measurement and BMI classification

Anthropometry was performed by trained personnel using standardised technique at the time of enrolment, before any dietary or pharmacological intervention. Body weight was measured to the nearest 0.1 kg on a calibrated digital platform scale, with the participant in light indoor clothing and without footwear, after voiding. Standing height was measured to the nearest 0.1 cm using a wall-mounted stadiometer, with the participant barefoot, heels together, buttocks and scapulae against the vertical board, and the head in the Frankfort horizontal plane. Each measurement was taken twice and the mean of the two readings was used; a third reading was obtained and the two closest values averaged if the first two differed by more than 0.5 kg or 0.5 cm. Instruments were calibrated against standard weights and a fixed reference length at the start of each recruitment month.

Body mass index was calculated as weight in kilograms divided by the square of height in metres (kg/m²). Participants were then assigned to BMI classes according to the standard classification of weight status by body mass index. Because Asian Indian body composition differs systematically from that of European-origin populations, the corresponding Asian-Indian consensus categories were recorded in parallel for every participant, so that the cohort could be described under both frameworks and so that the results would be comparable with both international and Indian literature. The dual classification scheme applied is set out in Table 2 in the Results section. For the primary association analysis, five BMI strata were used (underweight, normal weight, overweight, obesity class I and obesity class II or above), the highest categories being combined to avoid cells with very small expected counts.

Laboratory methods

Venous blood was collected at enrolment after an overnight fast of at least eight hours. Samples for

glycated haemoglobin were collected into di-potassium EDTA tubes, mixed gently by inversion, and processed on the day of collection; samples for plasma glucose were collected into sodium fluoride–potassium oxalate tubes and centrifuged within 30 minutes of venepuncture. HbA1c was estimated on an automated analyser using a method aligned to the National Glycohemoglobin Standardization Program and traceable to the IFCC reference measurement procedure, and results are reported in National Glycohemoglobin Standardization Program units (%) with IFCC units (mmol/mol) available by the standard master equation. HbA1c was therefore measured in whole blood collected into di-potassium EDTA tubes and not in serum; accordingly, the term "serum HbA1c" is not used in this report, because HbA1c is a glycated fraction of erythrocyte haemoglobin and cannot be measured in serum. Internal quality control at two levels was run with every analytical batch, and the laboratory participated in an external quality assurance programme throughout the study period; runs failing internal control limits were repeated. Where clinically indicated, HbA1c results were interpreted alongside a complete blood count and peripheral smear to exclude the haematological interferences listed in the exclusion criteria. Estimated average glucose values quoted for clinical interpretation were derived using the validated linear relationship between HbA1c and mean glucose. Fasting plasma glucose was measured by the glucose oxidase–peroxidase method on the same automated platform.

Variables

The exposure variable was BMI class, treated as a categorical variable with five ordered levels. The outcome variable was HbA1c at diagnosis, analysed both as a continuous variable (median and inter-quartile range) and, for the primary association test, as a categorical variable with five clinically anchored bands: <6.5% (diagnosis established by glucose criteria), 6.5–7.4% (mild hyperglycaemia, generally amenable to monotherapy), 7.5–8.9% (moderate hyperglycaemia, frequently requiring dual therapy), 9.0–9.9% (marked hyperglycaemia) and ≥10.0% (severe hyperglycaemia, in which early insulin or triple therapy is commonly indicated). These bands were chosen a priori to align with treatment-intensification thresholds in contemporary standards of care, so that the categories carry therapeutic and not merely statistical meaning. Descriptive covariates recorded at baseline included age, sex, height, weight, fasting plasma glucose, presenting symptoms, family history of diabetes in a first-degree relative, self-reported tobacco and alcohol use, and residence (urban, peri-urban or rural).

Data management and quality control

Data were recorded on structured case record forms, entered into a password-protected spreadsheet, and independently double-checked against source documents for a random 10% sample of records; all values for BMI and HbA1c were verified for every participant. Range and logic checks were applied to detect implausible entries (for example height <120 cm, weight <25 kg, BMI outside 12–60 kg/m², HbA1c outside 4–20%), and flagged records were re-verified against laboratory reports. Personal identifiers were replaced by study codes before analysis.

Statistical analysis

Analyses were performed on the complete baseline dataset of 410 participants. The dataset was analysed to assess the association between body mass index classes and glycated haemoglobin (HbA1c) levels. Categorical variables are presented as frequencies and percentages with 95% confidence intervals calculated by the Wilson score method. Because HbA1c and BMI in clinical populations are typically right-skewed rather than normally distributed, distributional assumptions were examined graphically and with the Shapiro–Wilk test. Continuous data are therefore presented as

median and inter-quartile range (IQR) with observed range; class-specific medians, IQRs and ranges, categorical composition, and Wilson confidence intervals for severe hyperglycaemia are displayed graphically.

The association between BMI class and HbA1c category was analysed with Pearson's chi-square test of independence applied to the 5 × 5 cross-tabulation, with degrees of freedom calculated as (rows − 1) × (columns − 1) = 16. Expected cell counts were inspected before interpretation to confirm that the test's assumptions were satisfied, and effect size was expressed as Cramér's V. Standardized (adjusted) residuals and each cell's percentage contribution to the total chi-square statistic were computed in order to identify which cells deviated most from independence; these cell-level quantities are presented graphically rather than tabulated, in order to avoid duplicating the same numbers in both a table and a figure. Supportive analyses treated HbA1c as a continuous variable and compared medians across BMI strata using the Kruskal–Wallis test, with the Jonckheere–Terpstra test used to look specifically for a monotonic trend across ordered BMI classes, since an ordered-alternative test is more sensitive than an omnibus test to the graded relationship predicted by the adiposity hypothesis. The monotonic and linear association between BMI and HbA1c on their original continuous scales was quantified by Spearman's rank correlation coefficient and by least-squares linear regression; the difference in the proportion of patients with HbA1c ≥ 9.0% across BMI classes was tested with a 2 × 5 chi-square test, with Wilson confidence intervals displayed graphically. A two-sided p value below 0.05 was considered statistically significant, and results are reported to the precision generated by the analysis software.

Ethical considerations

The parent trial and the present baseline analysis were conducted in accordance with the ethical principles of the Declaration of Helsinki and the National Ethical Guidelines for Biomedical and Health Research Involving Human Participants issued by the Indian Council of Medical Research [17,18]. Institutional Ethics Committee approval was obtained before recruitment, and the trial was prospectively registered with the Clinical Trials Registry – India (CTRI/2024/10/074542). Written informed consent was obtained from every participant in a language they understood, after explanation of the study purpose, procedures, risks and benefits, and of the right to withdraw at any time without effect on clinical care. No participant received a placebo in place of indicated therapy: all patients diagnosed with diabetes were started on guideline-directed treatment and lifestyle counselling immediately after baseline data collection. Data were stored in de-identified form and access was restricted to the investigators.

Results

Baseline characteristics of the study population

A total of 410 newly diagnosed, treatment-naïve patients with type 2 diabetes mellitus contributed complete anthropometric and HbA1c data and were included in the analysis. The baseline demographic, anthropometric, clinical and biochemical characteristics of the cohort are presented in Table 1. The cohort was middle-aged, with a slight male preponderance, and drew from urban, peri-urban and rural parts of the hospital catchment. A family history of diabetes in a first-degree relative was common, and a substantial proportion of patients were detected during evaluation of non-specific or unrelated complaints rather than through classical osmotic symptoms.

Table 1. Baseline demographic, anthropometric, clinical and biochemical characteristics of the study population (n = 410).

Characteristic	n (%) or ratio	95% CI (%)	Median (IQR)	Mean ± SD (95% CI)	Range
Demographic characteristics					
Age (years)	—	—	48 (41–56)	48.6 ± 10.8 (47.6–49.7)	24–78
Age group: ≤40 years	96 (23.4)	19.6–27.7	—	—	—
Age group: 41–60 years	247 (60.2)	55.4–64.9	—	—	—
Age group: >60 years	67 (16.3)	13.1–20.2	—	—	—
Sex: male	231 (56.3)	51.5–61.1	—	—	—
Sex: female	179 (43.7)	38.9–48.5	—	—	—
Male-to-female ratio	1.29:1	—	—	—	—
Residence: urban	171 (41.7)	37.0–46.5	—	—	—
Residence: peri-urban	108 (26.3)	22.3–30.8	—	—	—
Residence: rural	131 (32.0)	27.6–36.6	—	—	—
Anthropometric characteristics					
Height (cm)	—	—	162.0 (156.0–168.5)	—	143–184
Weight (kg)	—	—	66.5 (58.0–75.4)	—	38–112
Body mass index (kg/m²)	—	—	26.5 (22.8–30.8)	26.8 ± 5.5	14.7–43.4
BMI <25.0 kg/m²	162 (39.5)	34.9–44.3	—	—	—
BMI ≥23.0 kg/m² (Asian-Indian action point)	318 (77.6)	73.3–81.3	—	—	—
Glycaemic and biochemical characteristics					
Fasting plasma glucose (mg/dL)	—	—	168 (141–204)	181 ± 48 (176–186)	112–398
HbA1c (%, NGSP)	—	—	8.1 (7.0–9.3)	8.3 ± 1.7 (8.2–8.5)	5.7–14.1
HbA1c (mmol/mol, IFCC)	—	—	65 (53–78)	—	39–131
Estimated average glucose (mg/dL)	—	—	186 (154–220)	—	117–358
HbA1c ≥9.0%	140 (34.1)	29.7–38.9	—	—	—
HbA1c ≥10.0%	63 (15.4)	12.2–19.2	—	—	—
Clinical history and presentation					
Family history of diabetes	186 (45.4)	40.6–50.2	—	—	—
Presentation: classical osmotic symptoms	194 (47.3)	42.5–52.2	—	—	—
Presentation: incidental/unrelated evaluation	216 (52.7)	47.8–57.5	—	—	—
Current tobacco use	121 (29.5)	25.3–34.1	—	—	—

Data are n (%) unless otherwise stated. BMI, body mass index; CI, confidence interval; eAG, estimated average glucose; HbA1c, glycated haemoglobin; IFCC, International Federation of Clinical Chemistry; IQR, inter-quartile range; NGSP, National Glycohemoglobin Standardization Program; SD, standard deviation. Confidence intervals for proportions use the Wilson score method. Estimated average glucose was derived from HbA1c using the validated linear relationship.

Body mass index: distribution, classification and reclassification

BMI in the cohort ranged across the entire clinical spectrum, with a median of 26.5 kg/m² (IQR 22.8–30.8 kg/m²; observed range 14.7–43.4 kg/m²). Notably, the cohort was not predominantly obese by international criteria: 24 patients (5.9%) were underweight and 138 (33.7%) were of normal weight, so that 162 patients (39.5%) — nearly two in five newly diagnosed patients — would have been classified as "not overweight" by conventional cut-offs, while 126 patients (30.7%) met international criteria for obesity, of whom only 30 (7.3%) reached class II or above. Because the interpretation of any BMI-based analysis in an Indian cohort depends on the cut-offs adopted, the observed values within each class, the class frequencies and the corresponding Asian-Indian consensus categories are presented together in Table 2. Reclassification under Asian-Indian cut-offs moved 70 patients out of the normal-weight category and 52 patients out of the international overweight category into higher-risk categories, so that the proportion of patients below the overweight threshold fell from 39.5% (95% CI 34.9–44.3) to 22.4% (95% CI 18.7–26.7), while the proportion meeting a criterion for obesity rose from 30.7% to 60.5%. This reclassification is not a statistical curiosity: it changes the proportion of the cohort that a BMI-gated screening policy would ever have tested.

Table 2. Distribution of body mass index by international class, observed BMI values within each class, and the corresponding Asian-Indian consensus classification (n = 410).

International BMI class (kg/m²)	n (%)	95% CI (%)	Median BMI (IQR)	Mean ± SD	Observed range	Corresponding Asian-Indian category (cut-off, kg/m²)	n (%) under Asian-Indian criteria
Underweight (< 18.5)	24 (5.9)	4.0–8.6	17.4 (16.6–18.0)	17.1 ± 1.1	14.7–18.4	Underweight (< 18.5)	24 (5.9)
Normal weight (18.5–24.9)	138 (33.7)	29.3–38.4	22.4 (20.9–23.8)	22.2 ± 1.8	18.5–24.9	Normal (18.5–22.9) and at risk / overweight (23.0–24.9)	68 (16.6) and 70 (17.1)
Overweight (25.0–29.9)	122 (29.8)	25.5–34.4	27.1 (26.0–28.4)	27.2 ± 1.4	25.0–29.9	Obesity (≥ 25.0)	122 (29.8)
Obesity class I (30.0–34.9)	96 (23.4)	19.6–27.7	31.9 (30.8–33.2)	32.0 ± 1.4	30.0–34.8	Obesity, higher grade (≥ 30.0)	96 (23.4)
Obesity class II or above (≥ 35.0)	30 (7.3)	5.2–10.3	36.8 (35.6–38.4)	37.4 ± 2.2	35.0–43.4	Obesity, higher grade (≥ 30.0)	30 (7.3)
Whole cohort	**410 (100.0)**	—	**26.5 (22.8–30.8)**	**26.8 ± 5.5**	**14.7–43.4**	—	**410 (100.0)**
Below the overweight threshold	*162 (39.5)*	*34.9–44.3*	—	—	—	*Below the Asian-Indian overweight threshold*	*92 (22.4; 95% CI 18.7–26.7)*
Meeting a criterion for obesity	*126 (30.7)*	*26.5–35.4*	—	—	—	*Meeting the Asian-Indian obesity criterion*	*248 (60.5)*

CI, confidence interval (Wilson score method); IQR, inter-quartile range; SD, standard deviation. The right-hand columns map each international class onto the Asian-Indian consensus categories. All

subsequent analyses use the five international BMI strata so that the results remain directly comparable with the lifetime-risk literature; the Asian-Indian distribution is retained for interpretation in the discussion.

Distribution of glycated haemoglobin at diagnosis

HbA1c at the time of diagnosis was distributed widely and was shifted markedly to the right of the diagnostic threshold. The median HbA1c for the whole cohort was 8.1% (IQR 7.0–9.3%), equivalent to an estimated average glucose of approximately 186 mg/dL, and the distribution was right-skewed with a tail of severely hyperglycaemic patients. Only 46 patients (11.2%) had an HbA1c below 6.5% — that is, patients whose diabetes had been established on glucose criteria — whereas 140 patients (34.1%) presented with an HbA1c of 9.0% or above, and 63 patients (15.4%) presented with an HbA1c of 10.0% or above, a level at which early combination therapy or insulin is usually indicated. The full distribution is given in Table 3, and the glycaemic and anthropometric category distributions, together with the effect of the international and Asian-Indian BMI classification frameworks, are shown in Figure 1.

Table 3. Distribution of HbA1c at diagnosis across clinically anchored categories, with IFCC equivalents and estimated average glucose (n = 410).

HbA1c category (%, NGSP)	IFCC equivalent (mmol/mol)	Estimated average glucose (mg/dL)	n	% of cohort	95% CI (%)	Cumulative n	Cumulative %	Clinical interpretation
< 6.5	< 48	< 140	46	11.2	8.5–14.6	46	11.2	Diabetes established on glucose criteria; HbA1c below the diagnostic cut-off
6.5–7.4	48–57	140–166	96	23.4	19.6–27.7	142	34.6	Mild hyperglycaemia; monotherapy with lifestyle measures usually sufficient
7.5–8.9	58–74	169–209	128	31.2	26.9–35.9	270	65.9	Moderate hyperglycaemia; above target, dual therapy often required
9.0–9.9	75–85	212–237	77	18.8	15.3–22.8	347	84.6	Severe hyperglycaemia; early combination therapy indicated
≥ 10.0	≥ 86	≥ 240	63	15.4	12.2–19.2	410	100.0	Marked hyperglycaemia; insulin or triple therapy usually indicated
Total	—	—	**410**	**100.0**	—	**410**	**100.0**	Median 8.1% (65 mmol/mol); 34.1% at or above 9.0%

CI, confidence interval (Wilson score method); NGSP, National Glycohemoglobin Standardization Program; IFCC, International Federation of Clinical Chemistry.

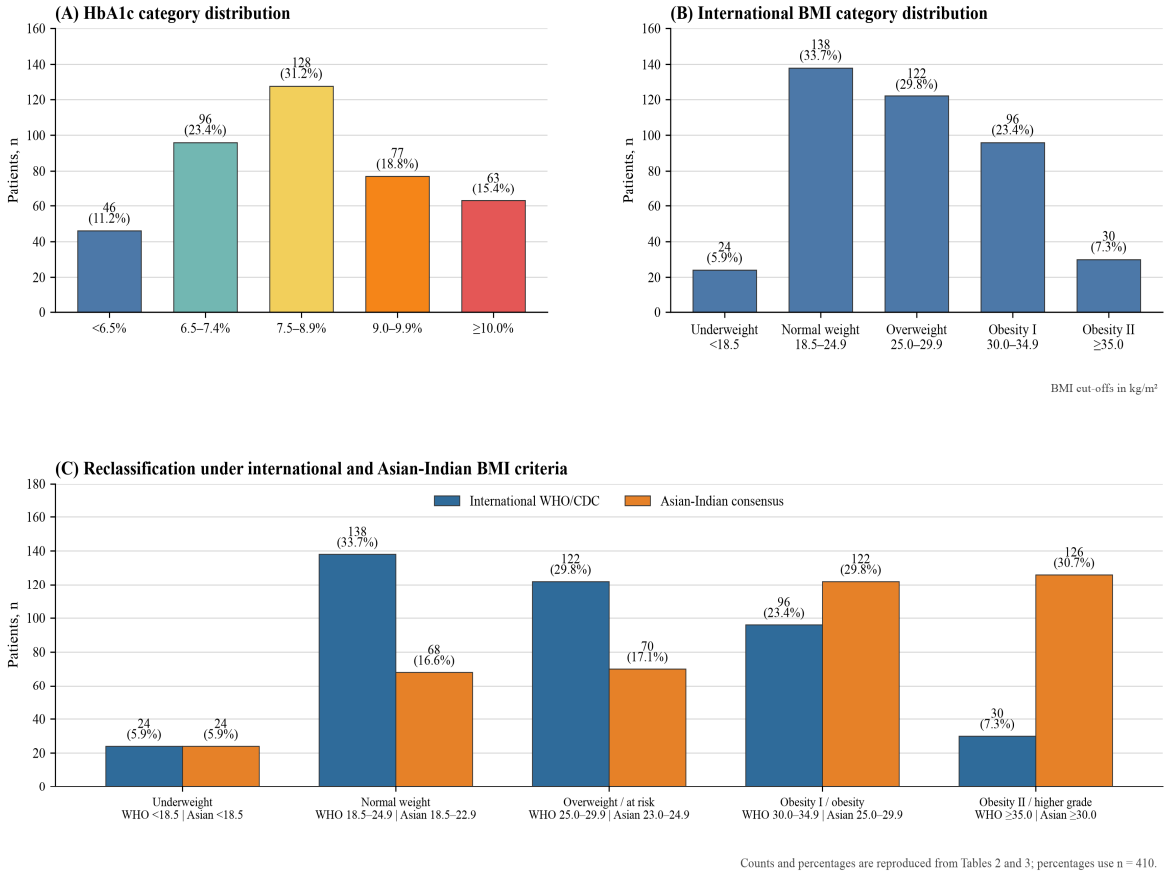

Figure 1. Glycaemic and anthropometric category profiles of the cohort at diagnosis (n = 410). **(A)** Distribution across the five HbA1c categories used in the analysis: <6.5%, 6.5–7.4%, 7.5–8.9%, 9.0–9.9% and ≥10.0%. Counts and percentages are shown above the bars. **(B)** Distribution across the five international WHO/CDC BMI classes: underweight (<18.5 kg/m²), normal weight (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), obesity class I (30.0–34.9 kg/m²) and obesity class II (≥35.0 kg/m²). **(C)** Reclassification of the same cohort using international WHO/CDC cut-offs (blue) and Asian-Indian consensus cut-offs (orange). The Asian-Indian thresholds reduce the normal-weight category and increase the number classified in obese categories. Percentages use n = 410. Values are reproduced directly from Tables 2 and 3.

Association between BMI class and HbA1c category

The central analysis cross-tabulated the five BMI classes against the five HbA1c bands; the observed counts and the corresponding within-class glycaemic summaries are presented together in Table 4. The distribution of HbA1c categories did not differ significantly across BMI classes (Pearson $\chi^2 = 14.3028$, df = 16, p = 0.576164). Expected cell counts satisfied Cochran's criteria for the test (mean expected count 16.4; the smallest expected count, in the underweight × HbA1c < 6.5% cell, was 2.7; five of the 25 cells, i.e. 20% of cells, had an expected count below 5, which meets the conventional tolerance of no more than 20% of cells below 5 with no expected count below 1). The corresponding effect size was small (Cramér's V = 0.093). Median HbA1c differed by no more than 0.9 percentage points between the extreme classes, and every class confidence interval overlapped the cohort interval.

Table 4. Cross-tabulation of BMI class against HbA1c category, with the glycaemic summary of each BMI class (n = 410).

BMI class (kg/m²)	n	HbA1c < 6.5%	6.5–7.4%	7.5–8.9%	9.0–9.9%	≥ 10.0%	Median HbA1c % (IQR)	95% CI of median	Mean ± SD (%)	Range (%)	Median mmol/mol	Median eAG (mg/dL)
Underweight (< 18.5)	24	1	5	7	5	6	8.9 (7.6–9.8)	7.6–9.3	9.2 ± 2.0	6.2–13.2	74	209
Normal weight (18.5–24.9)	138	17	33	44	26	18	8.0 (7.0–9.2)	7.6–8.5	8.2 ± 1.6	5.7–14.1	64	183
Overweight (25.0–29.9)	122	13	31	39	27	12	8.1 (7.0–9.1)	7.7–8.4	8.2 ± 1.5	5.7–13.6	65	186
Obesity class I (30.0–34.9)	96	12	23	28	15	18	8.0 (6.7–9.6)	7.6–8.7	8.4 ± 1.9	5.8–13.9	64	183
Obesity class II or above (≥ 35.0)	30	3	4	10	4	9	8.7 (7.8–10.0)	7.9–10.0	8.7 ± 1.6	5.9–12.5	72	203
Whole cohort	**410**	**46**	**96**	**128**	**77**	**63**	**8.1 (7.0–9.3)**	**7.9–8.4**	**8.3 ± 1.7**	**5.7–14.1**	**65**	**186**

IQR, inter-quartile range; SD, standard deviation; CI, confidence interval; eAG, estimated average glucose. Cell entries in the five HbA1c columns are patient counts; row totals equal the class size. Confidence intervals for medians are order-statistic (distribution-free) intervals. Pearson $\chi^2 = 14.3028$, df = 16, p = 0.576164; Cramér's V = 0.093. Within-class percentage composition and severe-hyperglycaemia confidence intervals are presented in Figure 2; expected counts, adjusted residuals and cell contributions to chi-square are presented in Figure 3.

The aggregate profiles underlying this result are displayed class by class in Figure 2. Median and IQR estimates overlap substantially, category compositions are similar, and the Wilson confidence intervals for the proportion with HbA1c ≥ 9.0% overlap across all five BMI classes. Supportive analyses treating HbA1c as a continuous variable were concordant: median HbA1c did not differ significantly across the five BMI strata (Kruskal–Wallis H = 7.75, df = 4, p = 0.101; $\varepsilon^2 = 0.009$), and no monotonic upward trend across ordered BMI classes was demonstrable (Jonckheere–Terpstra J = 31 124, z = 0.19, p = 0.849).

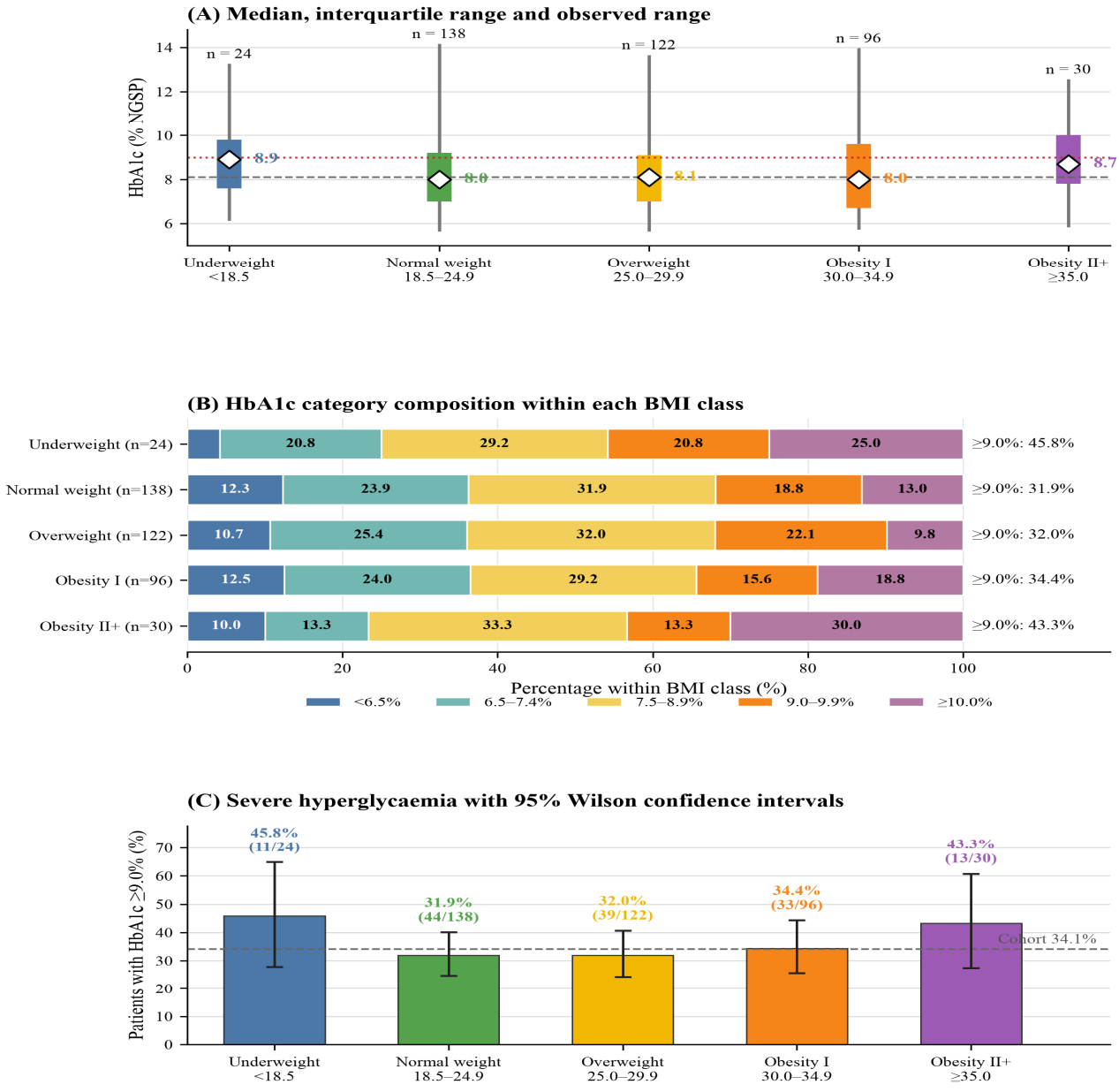

Figure 2. Aggregate glycaemic severity profiles across BMI classes (n = 410). **(A)** Class-specific median HbA1c (diamond), inter-quartile range (thick bar) and observed range (thin line), reproduced from Table 4. Dashed reference lines mark the cohort median (8.1%) and severe-hyperglycaemia threshold (9.0%). **(B)** Within-class composition across the five HbA1c bands; each horizontal bar totals 100%, and the proportion with HbA1c ≥9.0% is printed at right. **(C)** Proportion within each BMI class with HbA1c ≥9.0%, with numerators, denominators and 95% Wilson confidence intervals. The dashed line marks the cohort proportion (34.1%); the formal comparison was non-significant ($\chi^2 = 3.16$, df = 4, $p = 0.532$).

On the original continuous scales the two variables were essentially uncorrelated (Spearman $\rho = -0.002$, $p = 0.97$; Pearson $r = -0.007$), and least-squares regression yielded a slope of −0.002 HbA1c percentage points per unit of BMI ($R^2 = 0.0001$), that is, a predicted change of only 0.04 percentage points of HbA1c across the entire 20 kg/m² span of the cohort — a quantity without clinical meaning. To identify where, if

anywhere, the observed table departed from independence, expected counts and standardized residuals were computed for all 25 cells. Only two cells crossed the conventional threshold of ±1.96, both within the ≥ 10.0% column: a deficit of severely hyperglycaemic patients among the overweight (standardized residual −2.02) and an excess among patients with class II obesity or above (+2.31). Together these two cells accounted for 46.2% of the total chi-square statistic, yet the omnibus test remained far from significance, so they must be regarded as exploratory rather than as evidence of a real subgroup effect. The proportion of patients with HbA1c ≥ 9.0% likewise did not differ significantly across BMI classes (χ^2 = 3.16, df = 4, p = 0.532), although the point estimates followed a U-shaped pattern, being highest in the underweight (45.8%) and class II obese (43.3%) strata. The cell-level departures and their contributions to the omnibus chi-square statistic are shown in Figure 3; the continuous-scale correlation and regression analyses are summarised in Table 5.

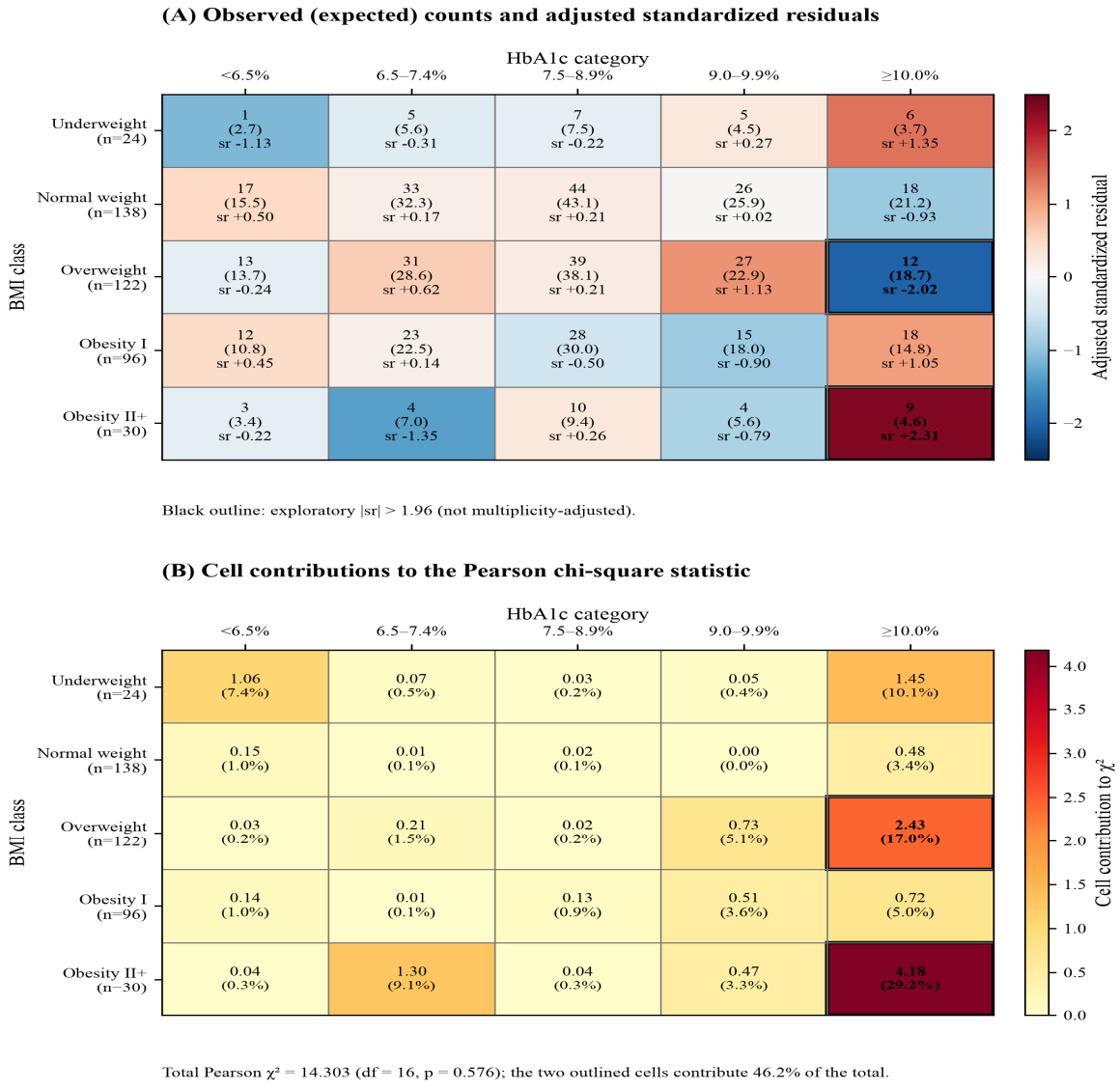

Figure 3. Cell-level structure of the BMI–HbA1c association (n = 410), reproduced from the observed 5 × 5 table in Table 4. **(A)** Each cell shows the observed count, expected count under independence in

parentheses and adjusted standardized residual (sr). Blue denotes fewer and red more patients than expected; the two cells with exploratory |sr| >1.96 are outlined. **(B)** Pearson chi-square contribution of each cell, with its percentage share of the total in parentheses. The two outlined cells—overweight × HbA1c ≥10.0% and obesity class II+ × HbA1c ≥10.0%—contribute 46.2% of the total χ^2, but the omnibus test is non-significant ($\chi^2 = 14.3028$, df = 16, p = 0.576164); cell-level findings are exploratory.

Summary of the statistical analysis

Table 5 consolidates the inferential output of the study, including the primary test, its degrees of freedom, the effect size, and the supportive analyses.

Table 5. Summary of all statistical analyses relating body mass index to glycated haemoglobin at diagnosis (n = 410).

Analysis	Test applied	Test statistic	df	p value	Effect size	Interpretation
Primary: BMI class (5) × HbA1c band (5)	Pearson chi-square test of independence	$\chi^2 = 14.3028$	16	0.576164	Cramér's V = 0.093	No statistically significant association; small effect size
Adequacy of the contingency analysis	Inspection of expected counts (Figure 3A)	Mean expected 16.4; minimum 2.7	—	—	—	5 of 25 cells (20%) with expected count < 5; minimum expected 2.7; Cochran's criteria met
Cell-level deviation from independence	Standardized residuals (Figure 3A)	Largest +2.31 (obesity II+ × ≥ 10.0%); smallest −2.02 (overweight × ≥ 10.0%)	—	—	46.2% of total χ^2 from 2 cells	2 of 25 cells exceed ±1.96; exploratory only
HbA1c (continuous) across 5 BMI classes	Kruskal–Wallis test	H = 7.75	4	0.101	$\varepsilon^2 = 0.009$	No difference in median HbA1c between classes
Ordered trend in HbA1c across BMI classes	Jonckheere–Terpstra test	J = 31 124; z = 0.19	—	0.849	—	No monotonic trend with increasing BMI
BMI versus HbA1c, both continuous	Spearman rank correlation	$\rho = -0.002$	408	0.97	$\rho^2 < 0.001$	No monotonic correlation
BMI versus HbA1c, both continuous	Pearson correlation and linear regression	r = −0.007; slope −0.002 %/unit BMI	408	0.89	$R^2 = 0.0001$	No linear relationship; slope clinically null
Severe hyperglycaemia (HbA1c ≥ 9.0%) across BMI classes	Pearson chi-square test (Figure 2C)	$\chi^2 = 3.16$	4	0.532	Cramér's V = 0.088	No statistically significant difference across BMI classes

df, degrees of freedom. All tests were two-sided with $\alpha = 0.05$. ε^2 is the epsilon-squared effect size for the Kruskal–Wallis test. Cramér's V is reported as an effect-size estimate and is interpreted in context rather than as variance explained.

Key observations

Four findings summarise the results. First, the burden of hyperglycaemia at diagnosis was high in every BMI class, with a cohort median HbA1c of 8.1% and 34.1% of patients at or above 9.0%. Second, BMI class carried no statistically detectable information about glycaemic severity, whether HbA1c was analysed as five ordered categories, as a continuous variable across five strata, as an ordered trend, or as a continuous correlate. Third, 39.5% of newly diagnosed patients were not overweight by international criteria and 5.9% were frankly underweight, so a screening strategy conditioned on obesity would have missed a large share of these patients — although under Asian-Indian cut-offs 77.6% of the cohort exceeded the 23.0 kg/m² action point. Fourth, if anything, the two extremes of the BMI distribution — underweight and class II obesity — carried the highest proportions of severe hyperglycaemia (45.8% and 43.3% with HbA1c ≥ 9.0%, respectively; Figure 2C), a U-shaped pattern that a linear adiposity model does not predict, although the small numbers in these strata mean this observation is hypothesis-generating rather than confirmatory.

Discussion

Among 410 newly diagnosed, treatment-naïve adults, HbA1c category was not significantly associated with BMI class ($\chi^2 = 14.3028$, df = 16, p = 0.576164; Cramér's V = 0.093). Glycaemic severity was high across the BMI spectrum: median HbA1c was 8.1%, 34.1% of participants had HbA1c ≥9.0%, and class-specific medians differed by less than one percentage point. At the same time, 39.5% of participants were not overweight and 5.9% were underweight by international criteria. The result therefore indicates that BMI class provided little information about the degree of hyperglycaemia at first diagnosis in this cohort.

This finding concerns severity at diagnosis rather than the probability of developing diabetes. BMI may strongly predict future diabetes in population cohorts while correlating weakly with HbA1c after disease is established.[19,20] Once patients are selected because they have crossed a diagnostic threshold, different combinations of insulin resistance, beta-cell failure, body composition, duration of undetected disease and recent catabolic weight loss may converge on similar HbA1c values.[21,22]

Several mechanisms may explain the weak association. BMI cannot distinguish fat from lean mass or identify visceral and ectopic fat.[23-28] Asian Indians may therefore have substantial metabolic adiposity despite a normal or low BMI, while some individuals with obesity retain greater beta-cell reserve. Direct measures such as waist circumference, body-fat percentage and visceral fat are likely to characterise metabolic risk more accurately than BMI alone.[29,30]

Early-life programming and beta-cell reserve may also be important. The thin-fat Indian phenotype combines relatively low lean mass with preserved central adiposity, and South Asian populations can develop beta-cell dysfunction at lower levels of BMI.[31,32] At the lean end, severe hyperglycaemia may reflect insulin deficiency or recent weight loss; at the obese end, it may reflect marked insulin resistance. The similar HbA1c burden across classes is therefore biologically plausible.[31-33]

The personal-fat-threshold model offers a complementary interpretation: diabetes develops when an individual exceeds their own safe storage capacity rather than a universal BMI cut-off. Patients can consequently reach a similar metabolic endpoint at very different body weights.[34,35] This framework also explains why non-obese diabetes should not automatically be regarded as mild disease.[36-38]

Clinically, BMI alone should not exclude adults from diabetes screening or determine the urgency of treatment. Presenting HbA1c, symptoms and catabolic features should guide initial therapy, and complication screening should begin at diagnosis irrespective of BMI. Ethnicity-specific anthropometric

thresholds remain useful, but they should complement rather than replace direct glycaemic assessment.[39,40]

Comparison with published literature

Published findings differ partly because studies address different outcomes. Western cohorts consistently show a strong relationship between BMI and diabetes incidence, whereas South Asian and body-composition studies show substantial metabolic risk at lower BMI. Studies of lean diabetes, glycaemic control and personal fat thresholds are more consistent with the weak BMI–severity relationship observed here. Representative evidence is summarised below.

Table 6. Comparison of the present study with representative published evidence; citation numbers identify each source.

Study / source	Population and design	Principal finding relevant to BMI–glycaemia	Relation to present study
Anjana et al. (ICMR–INDIAB) [2,3]	National Indian surveys	High prevalence with marked inter-state and urban–rural variation	Provides Indian epidemiological context
Narayan et al. [7]	US lifetime-risk modelling	Diabetes incidence rises sharply across BMI strata	Different outcome: incidence rather than severity
Hu et al.; Chan et al. [8,9]	Large US prospective cohorts	BMI, central adiposity and adult weight gain predict diabetes	Strong incidence relationship
Deurenberg et al.; Gallagher et al. [11,12]	Multi-ethnic body-composition studies	The BMI–body-fat relationship varies by ethnicity and age	Supports limitations of BMI
WHO Expert Consultation; Misra et al. [13,14]	Asian and Asian-Indian consensus guidance	Lower BMI and waist action points are appropriate for Asian populations	Supports ethnicity-specific classification
Garg and Dutta [19]	Indian cross-sectional study in underweight T2DM	Low BMI can coexist with adverse body composition	Concordant
Dudeja et al. [20]	Northern Indian body-composition study	BMI underestimated excess adiposity in Asian Indians	Supports BMI misclassification
Vikram et al. [21]	Northern Indian patients with T2DM	Percentage body fat identified excess adiposity not captured by BMI	Supports direct body-composition assessment
Misra [22]	Perspective on ethnicity-specific BMI criteria	Conventional BMI misclassifies metabolically at-risk Asian Indians	Concordant
Yajnik and colleagues [23,24]	Developmental and early-life evidence	The thin-fat Indian phenotype combines low lean mass with central adiposity	Mechanistically concordant
Ma and Chan; Unnikrishnan et al. [25,26]	Asian diabetes phenotype reviews	Beta-cell dysfunction can occur at lower adiposity	Mechanistically concordant
Vaag and Lund [27]	Review of non-obese T2DM	Non-obese T2DM may be a distinct insulinopenic phenotype	Concordant
George et al.; Coleman et al.; Gujral et al. [28–30]	Lean-diabetes cohorts and reviews	Lean and non-overweight diabetes is clinically important and may involve rapid beta-cell failure	Supports severe disease at low BMI
Bae et al. [31]	US electronic health-record analysis	Overweight and obesity were associated with poorer glycaemic goal attainment in treated diabetes	Different treated population and outcome
Taylor and Holman [32]	Mechanistic hypothesis	Personal fat threshold permits diabetes at normal BMI	Provides a unifying explanation

Study / source	Population and design	Principal finding relevant to BMI–glycaemia	Relation to present study
Rothman [33]	Methodological review	BMI can misclassify adiposity and introduce measurement error	Supports cautious interpretation
Present study	410 newly diagnosed, treatment-naïve Indian adults	No significant association between BMI class and HbA1c category	—

HbA1c interpretation is supported by established diagnostic guidance [34], the validated conversion to estimated average glucose [35], NGSP standardisation [36] and worldwide assay harmonisation [37]. Prognostic studies link higher glycaemia with microvascular and macrovascular complications [38], cardiovascular risk even below the diagnostic range [39], and higher cardiovascular and mortality risks with younger age at diabetes diagnosis [40]. Lifestyle intervention reduces progression to diabetes [41] and improves weight, fitness and glycaemic control in established disease [42].

Methodologically, the study used median, interquartile-range and box-plot summaries for skewed continuous data [43] and an established chi-square framework [44]. Additional strengths were the untreated cohort, standardised anthropometry, quality-assured HbA1c measurement, full-year recruitment, dual BMI classification and reporting of effect size alongside the p value.

Limitations

The cross-sectional design prevents causal or temporal inference, and BMI at diagnosis cannot capture previous weight trajectory or hyperglycaemia-related weight loss. This single-centre hospital cohort may be affected by referral and detection bias, limiting generalisability. Adiposity was assessed by BMI without waist circumference or direct body-composition measures; insulin, C-peptide, beta-cell function, insulin sensitivity and autoimmune markers were unavailable. Diet, physical activity, socio-economic status and duration of undiagnosed disease were not quantified. Categorising BMI and HbA1c reduced statistical information, although continuous and non-parametric analyses were concordant. The underweight and class II obesity groups were small, making apparent differences at the extremes imprecise. Finally, a non-significant result does not prove equivalence and cannot exclude a weak association.

Future multicentre prospective studies should combine treatment-naïve recruitment with waist measures, direct body composition, beta-cell and autoimmune markers, and longitudinal assessment of treatment response and complications.

Conclusion

The present study found that glycaemic severity at diagnosis was similar across BMI classes. In 410 newly diagnosed, treatment-naïve adults attending a tertiary care centre in Kanpur, HbA1c category was not associated with BMI class ($\chi^2 = 14.3028$; df = 16; p = 0.576164), the effect size was small, and median HbA1c varied by less than one percentage point across the BMI spectrum. Newly diagnosed diabetes was common in normal-weight and even underweight patients, and severe hyperglycaemia at presentation was as likely at the lean end of the distribution as at the obese end. Two practical messages follow. Clinically, BMI alone should not be used to exclude adults from diabetes screening, nor as a proxy for expected glycaemic severity or for the urgency of treatment intensification: the presenting HbA1c should drive therapy in every BMI class, and complication screening should begin at diagnosis for all patients. Scientifically, the finding reinforces the argument that in Asian Indians it is body-fat quantity and distribution, together with beta-cell reserve, and not body mass index, that shape the diabetes phenotype. Adopting ethnicity-

specific anthropometric criteria and incorporating direct measures of body composition into routine assessment are therefore likely to be more informative than continued reliance on BMI alone.

Declarations

Ethics approval and consent to participate: The parent randomised controlled trial (CTRI/2024/10/074542) and this baseline analysis were approved by the Institutional Ethics Committee of Rama Medical College Hospital and Research Centre, Kanpur. Written informed consent was obtained from all participants.

Availability of data and materials: The de-identified dataset is available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests.

Funding: No external funding was received for this analysis.

Authors' contributions: AMV conceived the analysis, collected and curated the data, performed the statistical analysis and drafted the manuscript. NN supervised the study design, methodology and pharmacological interpretation. KJ contributed to data collection and critical revision. SK provided clinical oversight of patient recruitment, diagnosis and management. All authors read and approved the final manuscript.

Acknowledgements: The authors thank the staff of the Departments of Pharmacology, General Medicine and Biochemistry, and above all the patients who consented to participate.

References

1. International Diabetes Federation. IDF Diabetes Atlas. 11th ed. Brussels, Belgium: International Diabetes Federation; 2025.
2. Anjana RM, Unnikrishnan R, Deepa M, Pradeepa R, Tandon N, Das AK, et al. Metabolic non-communicable disease health report of India: the ICMR-INDIAB national cross-sectional study (ICMR-INDIAB-17). Lancet Diabetes Endocrinol. 2023;11(7):474–89.
3. Anjana RM, Pradeepa R, Deepa M, Datta M, Sudha V, Unnikrishnan R, et al. Prevalence of diabetes and prediabetes (impaired fasting glucose and/or impaired glucose tolerance) in urban and rural India: phase I results of the Indian Council of Medical Research–INdia DIABetes (ICMR-INDIAB) study. Diabetologia. 2011;54(12):3022–7.
4. Mohan V, Sandeep S, Deepa R, Shah B, Varghese C. Epidemiology of type 2 diabetes: Indian scenario. Indian J Med Res. 2007;125(3):217–30.
5. Chan JCN, Malik V, Jia W, Kadowaki T, Yajnik CS, Yoon KH, et al. Diabetes in Asia: epidemiology, risk factors, and pathophysiology. JAMA. 2009;301(20):2129–40.
6. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser. 2000;894:i–xii, 1–253.
7. Narayan KMV, Boyle JP, Thompson TJ, Gregg EW, Williamson DF. Effect of BMI on lifetime risk for diabetes in the U.S. Diabetes Care. 2007;30(6):1562–6.
8. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, et al. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. N Engl J Med. 2001;345(11):790–7.
9. Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. Diabetes Care. 1994;17(9):961–9.
10. Kahn SE, Hull RL, Utzschneider KM. Mechanisms linking obesity to insulin

resistance and type 2 diabetes. Nature. 2006;444(7121):840–6.
11. Deurenberg P, Deurenberg-Yap M, Guricci S. Asians are different from Caucasians and from each other in their body mass index/body fat per cent relationship. Obes Rev. 2002;3(3):141–6.
12. Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR, Sakamoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. Am J Clin Nutr. 2000;72(3):694–701.
13. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004;363(9403):157–63.
14. Misra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, et al. Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. J Assoc Physicians India. 2009;57:163–70.
15. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Ann Intern Med. 2007;147(8):573–7.
16. American Diabetes Association Professional Practice Committee for Diabetes. 2. Diagnosis and classification of diabetes: Standards of Care in Diabetes—2026. Diabetes Care. 2026;49(Suppl 1):S27–49.
17. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human participants. JAMA. 2025;333(1):71–4.
18. Indian Council of Medical Research. National ethical guidelines for biomedical and health research involving human participants. New Delhi: Indian Council of Medical Research; 2017.
19. Garg DK, Dutta MK. Body mass composition among underweight type 2 diabetes mellitus patients — a cross-sectional comparative study. Indian J Endocrinol Metab. 2019;23(2):222–6.
20. Dudeja V, Misra A, Pandey RM, Devina G, Kumar G, Vikram NK. BMI does not accurately predict overweight in Asian Indians in northern India. Br J Nutr. 2001;86(1):105–12.
21. Vikram NK, Misra A, Pandey RM, Dudeja V, Sinha S, Ramadevi J, et al. Anthropometry and body composition in northern Asian Indian patients with type 2 diabetes: receiver operating characteristics curve analysis of body mass index with percentage body fat as standard. Diabetes Nutr Metab. 2003;16(1):32–40.
22. Misra A. Ethnic-specific criteria for classification of body mass index: a perspective for Asian Indians and American Diabetes Association position statement. Diabetes Technol Ther. 2015;17(9):667–71.
23. Yajnik CS, Yudkin JS. The Y-Y paradox. Lancet. 2004;363(9403):163.
24. Yajnik CS. Early life origins of insulin resistance and type 2 diabetes in India and other Asian countries. J Nutr. 2004;134(1):205–10.
25. Ma RCW, Chan JCN. Type 2 diabetes in East Asians: similarities and differences with populations in Europe and the United States. Ann N Y Acad Sci. 2013;1281(1):64–91.
26. Unnikrishnan R, Anjana RM, Mohan V. Diabetes in South Asians: is the phenotype different? Diabetes. 2014;63(1):53–5.
27. Vaag A, Lund SS. Non-obese patients with type 2 diabetes and prediabetic subjects: distinct phenotypes requiring special diabetes treatment and (or) prevention? Appl Physiol Nutr Metab. 2007;32(5):912–20.

28. George AM, Jacob AG, Fogelfeld L. Lean diabetes mellitus: an emerging entity in the era of obesity. World J Diabetes. 2015;6(4):613–20.
29. Coleman NJ, Miernik J, Philipson L, Fogelfeld L. Lean versus obese diabetes mellitus patients in the United States minority population. J Diabetes Complications. 2014;28(4):500–5.
30. Gujral UP, Weber MB, Staimez LR, Narayan KMV. Diabetes among non-overweight individuals: an emerging public health challenge. Curr Diab Rep. 2018;18(8):60.
31. Bae JP, Lage MJ, Mo D, Nelson DR, Hoogwerf BJ. Obesity and glycemic control in patients with diabetes mellitus: analysis of physician electronic health records in the US from 2009–2011. J Diabetes Complications. 2016;30(2):212–20.
32. Taylor R, Holman RR. Normal weight individuals who develop type 2 diabetes: the personal fat threshold. Clin Sci (Lond). 2015;128(7):405–10.
33. Rothman KJ. BMI-related errors in the measurement of obesity. Int J Obes (Lond). 2008;32(Suppl 3):S56–9.
34. World Health Organization. Use of glycated haemoglobin (HbA1c) in the diagnosis of diabetes mellitus: abbreviated report of a WHO consultation. Geneva: World Health Organization; 2011.
35. Nathan DM, Kuenen J, Borg R, Zheng H, Schoenfeld D, Heine RJ; A1c-Derived Average Glucose Study Group. Translating the A1C assay into estimated average glucose values. Diabetes Care. 2008;31(8):1473–8.
36. Little RR, Rohlfing CL, Sacks DB; National Glycohemoglobin Standardization Program Steering Committee. Status of hemoglobin A1c measurement and goals for improvement: from chaos to order for improving diabetes care. Clin Chem. 2011;57(2):205–14.
37. Consensus Committee. Consensus statement on the worldwide standardization of the hemoglobin A1c measurement. Diabetes Care. 2007;30(9):2399–400.
38. Stratton IM, Adler AI, Neil HAW, Matthews DR, Manley SE, Cull CA, et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. BMJ. 2000;321(7258):405–12.
39. Selvin E, Steffes MW, Zhu H, Matsushita K, Wagenknecht L, Pankow J, et al. Glycated hemoglobin, diabetes, and cardiovascular risk in nondiabetic adults. N Engl J Med. 2010;362(9):800–11.
40. Sattar N, Rawshani A, Franzén S, Rawshani A, Svensson AM, Rosengren A, et al. Age at diagnosis of type 2 diabetes mellitus and associations with cardiovascular and mortality risks. Circulation. 2019;139(19):2228–37.
41. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al.; Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med. 2002;346(6):393–403.
42. Look AHEAD Research Group; Wing RR, Bolin P, Brancati FL, Bray GA, Clark JM, Coday M, et al. Cardiovascular effects of intensive lifestyle intervention in type 2 diabetes. N Engl J Med. 2013;369(2):145–54.
43. Krzywinski M, Altman N. Visualizing samples with box plots. Nat Methods. 2014;11(2):119–20.
44. McHugh ML. The chi-square test of independence. Biochem Med (Zagreb). 2013;23(2):143–9.