ORIGINAL ARTICLE

Real-world Experience with Tirzepatide (Mounjaro/Yurpeak) in Indian Patients with Type 2 Diabetes and Obesity: A Multicenter Retrospective Observational Study

Rikin Shah[1], Sanjeev Phatak[2], Mayur Agrawal[3], Chirag Aghara[4], Rucha Mehta[5], Dhruvi Hasnani[6], Vipul Chavda[7]

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Abstract

Introduction: Obesity and type 2 diabetes mellitus (T2DM) represent an escalating dual epidemic in India, compounded by a unique South Asian metabolic phenotype characterized by early-onset insulin resistance, visceral adiposity, and elevated cardiometabolic risk at lower body mass index (BMI) thresholds. Tirzepatide, a first-in-class dual glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1) receptor agonist, has demonstrated superior efficacy in global phase 3 trials. Real-world Indian data remain limited.

Materials and methods: This multicenter, retrospective observational study enrolled 331 adult Indian patients with obesity and/or T2DM who initiated once-weekly subcutaneous tirzepatide (starting dose, 2.5 mg, titrated to maximum tolerated dose) across routine outpatient clinics (December 2025 to January 2026). Primary outcomes were absolute and percentage weight loss. Secondary outcomes included HbA1c reduction, weight-loss responder rates, tolerability, and patient satisfaction. Wilcoxon signed-rank tests assessed paired comparisons; Kruskal–Wallis and Mann–Whitney *U* tests compared intergroup differences ($\alpha = 0.05$, two-tailed).

Results: A total of 331 patients (mean age, 45.1 ± 13.1 years; 50.2% male; T2DM, 47.1%; prediabetes, 11.2%; nondiabetic obesity, 41.7%) were included. Mean baseline weight was 98.2 ± 19.1 kg and BMI was 36.1 ± 6.2 kg/m^2. Mean weight loss was 6.67 ± 5.03 kg (6.76% body weight; Wilcoxon W = 739.5, $p < 0.0001$); 54.0 and 21.3% achieved ≥5 and ≥10% weight loss, respectively. Among patients with paired HbA1c data ($n = 137$), mean HbA1c decreased from 8.05 ± 1.93% to 6.94 ± 1.20% (reduction, 1.11 ± 1.21%; $p < 0.0001$), with 65.7% achieving HbA1c <7.0%. Nondiabetic patients lost significantly more weight than T2DM patients (7.9 vs 5.4 kg; Mann–Whitney $U = 7,760$, $p = 0.0001$). A clear dose-response relationship was observed (Spearman $\rho = 0.47$; $p < 0.0001$). Gastrointestinal adverse events were reported in 89.4% (predominantly mild to moderate); treatment discontinuation occurred in 16.9%; high patient satisfaction was reported by 46.2%.

Conclusion: Tirzepatide demonstrates clinically meaningful early weight reduction and glycemic improvement in Indian adults with T2DM and/or obesity in routine clinical practice, with a clear dose-response relationship and an acceptable tolerability profile. These findings represent the first large real-world Indian multicenter experience with tirzepatide, supporting its role as a valuable metabolic therapeutic in Indian clinical practice.

Keywords: Dual glucose-dependent insulinotropic polypeptide/glucagon-like peptide-1 receptor agonist, Glycemic control, Obesity, Real-world evidence, Tirzepatide, Type 2 diabetes mellitus, Weight loss.

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Introduction

Obesity is recognized as a serious chronic, complex disease by the World Health Organization, and its prevalence is rapidly escalating in India. After the United States and China, India ranks 3rd globally in the number of people living with obesity.[1] Between 1990 and 2022, the prevalence of obesity increased from 1.2 to 9.8% in women and from 0.5 to 5.4% in men.[2] According to the ICMR-INDIAB national study, there are 254 million and 351 million adults with generalized and abdominal obesity, respectively.[3]

The Indian metabolic phenotype is characterized by unique risk factors compounding this burden. For any given body mass index (BMI), Asians have approximately 3–5% higher adiposity or visceral fat compared to Caucasians.[4] Revised NICE 2025 guidelines recommend lower BMI thresholds for South Asians: overweight ≥23 kg/m^2 and obesity ≥25 kg/m^2.[5,6] Additional features include a high prevalence of central obesity, sarcopenic obesity, insulin resistance, atherogenic dyslipidemia, carbohydrate-rich dietary patterns, and type 2 diabetes mellitus (T2DM) onset nearly two decades earlier than in Western populations.[7–12]

Tirzepatide (Mounjaro®, Eli Lilly), the first-in-class dual glucose-dependent insulinotropic polypeptide (GIP)/glucagon-like peptide-1 (GLP-1) receptor agonist, operates *via* an imbalanced and biased dual receptor pharmacology, preferentially engaging the GIP receptor

[1]Re Life Clinic—Diabetes and Obesity Centre, Ahmedabad, Gujarat, India
[2]Vijayratna Diabetes and Obesity Centre, Ahmedabad, Gujarat, India
[3]Hormone India Diabetes and Endocrine Centre, Bhopal, Madhya Pradesh, India
[4]Uma Diabetes Center, Morbi, Gujarat, India
[5]EDMO Health Solutions, Ahmedabad, Gujarat, India
[6,7]Rudraksha Institute of Medical Sciences, Ahmedabad, Gujarat, India

Corresponding Author: Rikin Shah, Re Life Clinic—Diabetes and Obesity Centre, Ahmedabad, Gujarat, India, Phone: +91 8866793437, e-mail: rkn.shah.m@gmail.com

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while exhibiting β-arrestin-biased agonism at the GLP-1 receptor, resulting in concurrent improvements in β-cell function, insulin sensitivity, and glucagon suppression superior to selective GLP-1 receptor agonists.[13] The SURPASS phase 3 program demonstrated HbA1c reductions of 1.87–2.58% and body weight reductions of 7.0–11.7 kg.[14,15] The SURMOUNT program demonstrated 14.7–20.9% weight reduction in adults with obesity.[16,17] The SURMOUNT-CN trial in Chinese adults showed weight reductions of 13.6–17.5% at 52 weeks.[18]

Network meta-analyses across 28–76 RCTs consistently confirm tirzepatide 15 mg as the most efficacious agent for both HbA1c reduction (SUCRA 94.2%; high certainty) and weight loss among all approved incretin therapies.[19,20] Despite this strong evidence base, real-world Indian data on tirzepatide remain limited. We conducted the first large multicenter retrospective observational study evaluating tirzepatide in Indian adults with T2DM and/or obesity in routine clinical practice.

Materials and Methods

Study Design and Data Source

This was a multicenter, retrospective, observational study conducted across multiple diabetes and obesity outpatient clinics in urban India. Data were extracted from structured electronic medical record databases (December 2025 to January 2026). The study was conducted in accordance with the Declaration of Helsinki. Informed consent was waived because of the retrospective, deidentified nature of the data.

Study Population

Adult patients (age ≥18 years) with obesity (BMI ≥25 kg/m^2 by Indian cutoffs) and/or T2DM or prediabetes who were initiated on once-weekly subcutaneous tirzepatide in routine clinical care and had documented baseline and at least one follow-up body weight measurement were eligible. A total of 355 responses were collected; 24 duplicate records were excluded, yielding 331 unique patients. Follow-up duration ranged from <4 weeks to 40 weeks, with a median of approximately 10 weeks (IQR, 6–16 weeks).

Treatment Protocol

Tirzepatide was initiated at 2.5 mg subcutaneously once weekly (94.6% of patients), consistent with the approved dose-escalation protocol. Dose escalation was performed in 2.5 mg increments at ≥4-week intervals based on tolerability and clinical response, up to a maximum of 15 mg weekly.

Outcomes

Primary: Absolute and percentage change in body weight from baseline to follow-up.

Secondary: HbA1c reduction (paired data); BMI change; waist circumference change; weight-loss responder rates (≥5%, ≥10%, ≥15%, ≥20%).

Safety: GI adverse event incidence and severity; treatment discontinuation; patient-reported satisfaction.

Statistical Analysis

Continuous variables are reported as mean ± standard deviation (SD) or median (IQR). Categorical variables are reported as counts (%). Paired comparisons used the Wilcoxon signed-rank test (nonparametric; nonnormal distribution of weight loss values). Intergroup comparisons used the Kruskal–Wallis H test (≥3 groups) or Mann–Whitney *U* test (two groups). Correlations used Spearman ρ. Categorical associations used the Chi-squared test.

Data on gastrointestinal adverse events were captured via a structured physician-administered checklist at each clinic visit, categorizing severity as none, mild (tolerable, no dose modification required), moderate (causing discomfort, dose modification considered), or severe (intolerable, requiring treatment discontinuation). Patient satisfaction was assessed using a single-item global satisfaction rating recorded by the treating physician at follow-up (high/moderate/low), reflecting the patient's self-reported overall experience with tirzepatide therapy. This instrument was not a formally validated psychometric tool; findings should therefore be interpreted as indicative rather than definitive.

Study data were collected via a structured electronic data-capture form completed by participating clinicians from their clinic records; the 355 initial responses represent individual patient data entries submitted by treating physicians, of which 24 duplicate records were identified and excluded, yielding 331 unique patients for analysis.

All tests were two-tailed; $\alpha = 0.05$. Analyses were conducted on available-case data without imputation using SPSS (version 31.0.1.0). Continuous variables are reported as mean ± standard deviation (SD) or median [interquartile range (IQR)]. Categorical variables are reported as counts (%). Paired within-patient comparisons were assessed using the Wilcoxon signed-rank test. Intergroup comparisons used the Kruskal–Wallis H test (≥3 groups) or Mann–Whitney *U* test (two groups). Correlations used Spearman rank correlation (ρ). Categorical associations used the Chi-squared test. All tests were two-tailed; $\alpha = 0.05$. Analyses were performed on available-case data without imputation using Python 3.11 (pandas, scipy, statsmodels) (Fig. 1).

Results

Study Population and Baseline Characteristics

A total of 331 adult Indian patients were included after exclusion of 24 duplicate records from 355 submitted responses. Mean age was 45.1 ± 13.1 years (range, 17–86 years); 166 (50.2%) were male and 164 (49.5%) were female. By glycemic status: T2DM, 156 (47.1%); prediabetes, 37 (11.2%); and nondiabetic obesity, 138 (41.7%). Baseline weight was 98.2 ± 19.1 kg and BMI was 36.1 ± 6.2 kg/m^2. The majority were initiated on 2.5 mg tirzepatide (94.6%). Baseline characteristics are summarized in Table 1.

Primary Outcome: Body Weight

Statistically highly significant weight reductions were observed at follow-up (Wilcoxon signed-rank W = 739.5, $p = 9.1 \times 10^{-52}$). Mean body weight decreased from 98.2 ± 19.1 kg to 91.5 ± 18.4 kg, an absolute reduction of 6.67 ± 5.03 kg [6.76 ± 4.78% body weight; median, 5.0 kg (IQR, 3.0–9.0 kg)]. Changes in anthropometric and glycemic parameters are summarized in Table 2.

Weight-loss responder analysis demonstrated that 54.0% of patients achieved ≥5% weight loss, 21.3% achieved ≥10%, 7.6% achieved ≥15%, and 3.0% achieved ≥20% weight loss. Responder rates are detailed in Table 3.

Nondiabetic patients achieved significantly greater weight loss than T2DM patients (7.90 ± 5.37 kg vs 5.41 ± 3.92 kg; Mann–Whitney $U = 7{,}760$, $p = 0.0001$), with prediabetes intermediate (7.32 ± 6.56 kg; Kruskal–Wallis H = 15.65, $p = 0.0004$). Weight loss by glycemic group is summarized in Table 4 and Figure 2.

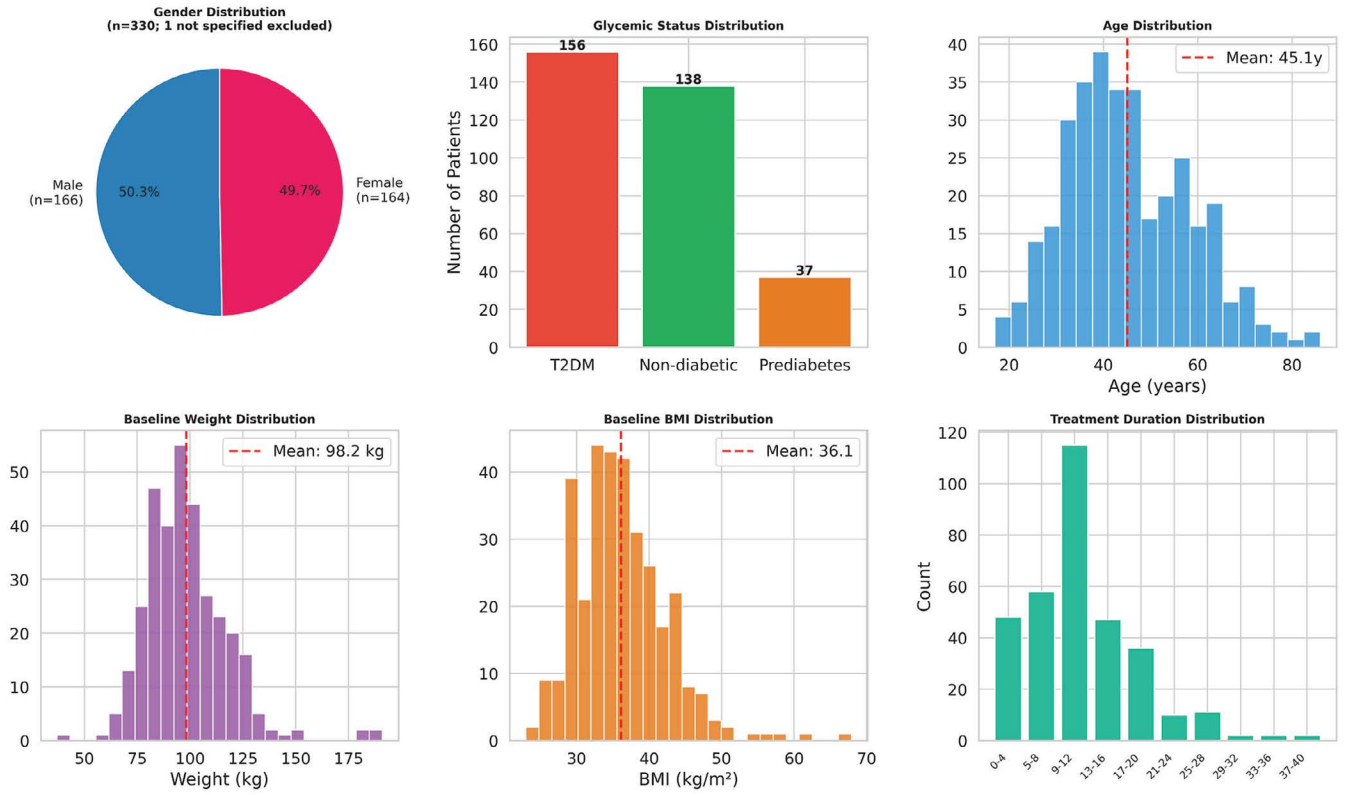

Fig. 1: Patient characteristics (*N* = 331). Distribution of gender, glycemic status, age, baseline weight, BMI, and treatment duration across the study cohort

Table 1: Baseline demographic and cardiometabolic characteristics (*N* = 331)

Variable	*Overall (N = 331)*	*T2DM/non-T2DM*
Demographic characteristics		
Total patients	331	156/175
Age (years), mean ± SD	45.1 ± 13.1	52.3 ± 13.2/38.6 ± 10.1
Age range (years)	188–86	–
Male, *n* (%)	166 (50.2%)	82 (52.6%)/84 (48.0%)
Female, *n* (%)	164 (49.5%)	74 (47.4%)/90 (51.4%)
Anthropometric parameters		
Baseline weight (kg), mean ± SD	98.2 ± 19.1	95.8 ± 18.4/100.3 ± 19.6
Baseline BMI (kg/m^2), mean ± SD	36.1 ± 6.2	35.0 ± 6.1/37.1 ± 6.2
Baseline waist circumference (cm)*	107.8 ± 22.0 (*n* = 157)	–
Glycemic status		
T2DM, *n* (%)	156 (47.1%)	–
Prediabetes, *n* (%)	37 (11.2%)	–
Nondiabetic (obesity), *n* (%)	138 (41.7%)	–
Baseline HbA1c (%), mean ± SD**	7.53 ± 1.99 (*n* = 205)	T2DM: 8.38 ± 1.91
Treatment characteristics		
Initial dose 2.5 mg, *n* (%)	313 (94.6%)	–
Prior OAD therapy, *n* (%)	107 (32.3%)	–
Prior insulin use, *n* (%)	25 (7.6%)	–
Follow-up ≤12 weeks, *n* (%)	198 (59.8%)	–
Follow-up 13–24 weeks, *n* (%)	97 (29.3%)	–
Follow-up >24 weeks, *n* (%)	36 (10.9%)	–
Maximum follow-up duration	40 weeks	–

*Waist circumference available in *n* = 157. **HbA1c available in diabetic/prediabetic patients only. BMI, body mass index; HbA1c, glycated hemoglobin; OAD, oral antidiabetic drug; SD, standard deviation. Gender data available for 330/331 patients; one patient's gender was not recorded

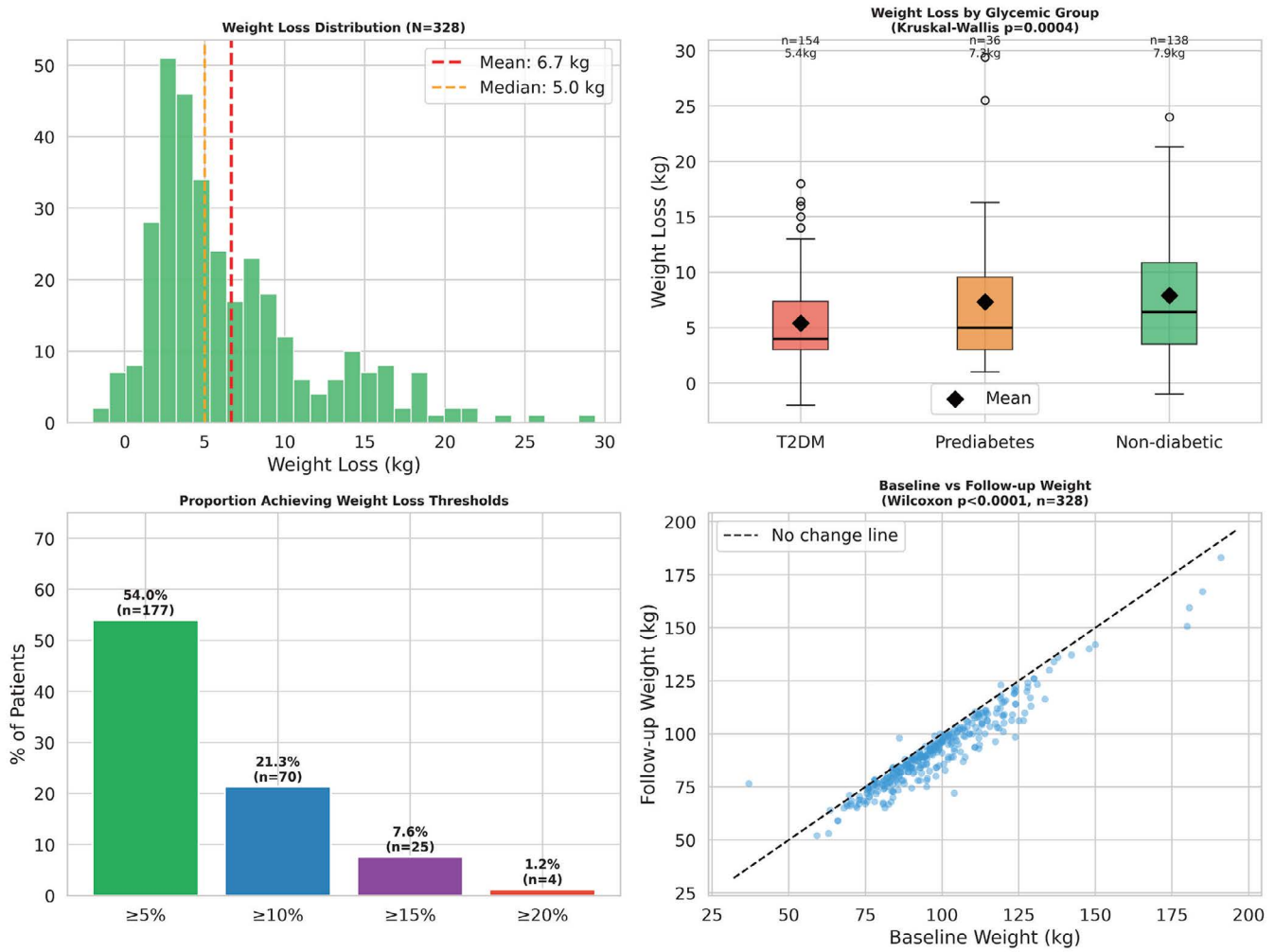

Fig. 2: Weight loss outcomes. (A) Weight loss distribution ($N = 328$; mean = 6.7 kg, median = 5.0 kg); (B) Weight loss by glycemic group (Kruskal–Wallis $p = 0.0004$; ♦ = mean); (C) Proportion of patients achieving weight loss thresholds; (D) Baseline vs follow-up weight scatter plot (Wilcoxon $p < 0.0001$, $n = 328$). Dashed line = line of no change

Table 2: Changes in anthropometric and glycemic parameters from baseline to follow-up

Parameter	*N*	*Baseline (mean ± SD)*	*Follow-up (mean ± SD)*	*Change (mean ± SD)*	*p-value*
Weight outcomes					
Weight (kg)	323	98.2 ± 19.1	91.5 ± 18.4	−6.67 ± 5.03	<0.001
BMI (kg/m^2)	323	36.1 ± 6.2	33.5 ± 6.0	−2.60 ± 2.12	<0.001
% weight loss	328	–	–	6.76 ± 4.78%	<0.001
Median weight loss (IQR)	328	–	–	5.0 (3.0–9.0) kg	<0.001
HbA1c outcomes (paired data)					
HbA1c (%)	137	8.05 ± 1.93	6.94 ± 1.20	−1.11 ± 1.21%	<0.001
T2DM subgroup HbA1c	96	8.56 ± 1.88	6.90 ± 1.10	−1.66 ± 1.29%	<0.001
Prediabetes HbA1c	11	6.28 ± 0.44	5.94 ± 0.44	−0.34 ± 0.36%	<0.001

BMI, body mass index; ETD, estimated treatment difference; HbA1c, glycated hemoglobin; IQR, interquartile range; SD, standard deviation. Wilcoxon signed-rank test used for all paired comparisons. Weight (kg) row: "$N = 323$; eight patients excluded due to missing follow-up weight." % Weight loss row: "$N = 328$; three patients excluded due to missing follow-up weight"

Dose–Response Relationship

A statistically significant dose–response relationship was observed across maximum tolerated dose categories (Spearman $\rho = 0.47$, $p < 0.0001$). Mean weight loss increased progressively from 3.11 ± 4.07 kg at 2.5 mg to 13.97 ± 3.81 kg at 15 mg. The proportion achieving ≥10% weight loss increased from 32.1% at 2.5 mg to 100% at 15 mg. Dose-response data are presented in Table 5 and Figure 3.

Glycemic Outcomes

Among patients with paired HbA1c data (n = 137), mean HbA1c decreased significantly from 8.05 ± 1.93% at baseline to 6.94 ± 1.20% at follow-up (reduction, 1.11 ± 1.21%; Wilcoxon W = 170.5, $p = 1.05 \times 10^{-20}$). In the T2DM subgroup (n = 96), mean HbA1c reduction was 1.43 ± 1.29%. At follow-up, 65.7% achieved HbA1c <7.0%, 57.7% achieved <6.5%, and 22.6% achieved normoglycemia (<5.7%). HbA1c outcomes stratified by baseline glycemic category are presented in Table 6 and Figure 4.

The left panel shows the distribution of HbA1c reduction, with a mean decrease of 1.11% (dashed line), indicating overall glycemic improvement across the cohort. The central panel demonstrates paired HbA1c values at baseline and follow-up, showing a significant reduction from 8.05% ± 1.93 to 6.94% ± 1.20 (Wilcoxon $p < 0.0001$). Individual patient trajectories (gray lines) highlight consistent downward trends. The right panel illustrates the proportion of patients achieving glycemic targets at follow-up: 65.7% achieved HbA1c <7.0%, 57.7% achieved <6.5%, and 22.6% achieved <5.7%.

Table 3: Weight-loss responder rates—proportion of patients achieving predefined thresholds (N = 328)

Weight loss threshold	*N achieving*	*Proportion of patients*
≥5% body weight reduction	177/328	54.0%
≥10% body weight reduction	70/328	21.3%
≥15% body weight reduction	25/328	7.6%
≥20% body weight reduction	10/328	3.0%

Wilcoxon signed-rank test; $p < 0.0001$ for all thresholds

Safety, Tolerability, and Patient Satisfaction

Gastrointestinal adverse events were reported in 296 (89.4%) patients: none in 10.6%, mild in 44.7%, moderate in 42.3%, and severe in 2.4%. GI event rates did not differ significantly across glycemic groups (Chi-square $p = 0.42$). Treatment discontinuation occurred in 56 patients (16.9%); the prediabetes group had the highest rate (27.0%, n = 10), followed by T2DM (17.9%, n = 28) and nondiabetic patients (13.0%, n = 18). Patient satisfaction was high in 46.2%, moderate in 40.8%, and low in 13.0%. Nondiabetic patients reported significantly higher satisfaction than T2DM patients (60.9 vs 30.8%; Chi-square $p < 0.0001$). Safety and satisfaction data are summarized in Table 7 and Figure 5.

Predictors of Weight Loss

Spearman correlation analyses identified treatment duration ($\rho = 0.592$, $p < 0.0001$) and baseline weight ($\rho = 0.238$, $p < 0.0001$) as significant positive predictors of absolute weight loss (Fig. 6). Longer treatment duration demonstrated a stronger association, consistent with the progressive weight-loss kinetics of tirzepatide observed across the SURPASS and SURMOUNT programs.

Table 4: Weight loss by glycemic group and responder analysis

Group	*N*	*Mean weight loss (kg ± SD)*	*% body weight lost*	*≥10% weight loss, n (%)*
T2DM	154	5.41 ± 3.92	5.73%	17/154 (11.0%)
Prediabetes	36	7.32 ± 6.56	6.68%	10/36 (27.8%)
Nondiabetic (obesity)	138	7.90 ± 5.37	7.92%	43/138 (31.2%)
Overall	328	6.67 ± 5.03	6.76%	70/328 (21.3%)

Kruskal–Wallis H = 15.65, $p = 0.0004$. Mann–Whitney U (T2DM vs nondiabetic): U = 7,760, $p = 0.0001$. SD, standard deviation. N = 154 for the T2DM subgroup; Two patients excluded due to missing follow-up weight data

Table 5: Dose-response—weight loss by maximum tolerated dose of tirzepatide

Max dose (mg)	*N (%)*	*Mean weight loss (kg ± SD)*	*% body weight lost*	*≥10% weight loss, n (%)*
2.5	28 (8.5%)	3.11 ± 4.07	3.11%	9 (32.1%)
5.0	165 (49.8%)	4.89 ± 3.18	5.20%	78 (47.3%)
7.5	67 (20.2%)	7.33 ± 4.26	7.75%	43 (64.2%)
10.0	46 (13.9%)	10.46 ± 6.16	9.96%	35 (76.1%)
12.5	13 (3.9%)	12.62 ± 6.49	12.21%	11 (84.6%)
15.0	12 (3.6%)	13.97 ± 3.81	12.33%	12 (100%)

Spearman $\rho = 0.47$ ($p < 0.0001$) for correlation between maximum dose and weight loss. Presented descriptively; no formal interdose statistical comparison performed. SD, standard deviation

Table 6: HbA1c reduction stratified by baseline HbA1c category (n = 137)

Baseline HbA1c category	*N*	*Baseline HbA1c (mean ± SD)*	*Follow-up HbA1c (mean ± SD)*	*p-value*	*Change (mean ± SD)*
<7.0%	52	6.41 ± 0.29	5.71 ± 0.44	<0.001	−0.70 ± 0.43%
7.0–9.0%	71	7.76 ± 0.56	6.72 ± 0.71	<0.001	−1.04 ± 0.72%
>9.0%	14	10.21 ± 1.10	7.95 ± 1.03	0.321	−2.26 ± 1.38%
Overall	137	8.05 ± 1.93	6.94 ± 1.20	<0.001	−1.11 ± 1.21%

ETD, estimated treatment difference; HbA1c, glycated hemoglobin; SD, standard deviation. All categories $p < 0.001$ by Wilcoxon signed-rank test except >9.0% (small n, $p = 0.321$)

Table 7: Safety, tolerability, and patient satisfaction by glycemic group

Variable	*Overall (N = 331)*	*T2DM (n = 156)*	*Nondiabetic (n = 138)*
Gastrointestinal adverse events			
Any GI event, *n* (%)	296 (89.4%)	143 (91.7%)	120 (87.0%)
None, *n* (%)	35 (10.6%)	13 (8.3%)	18 (13.0%)
Mild, *n* (%)	148 (44.7%)	70 (44.9%)	62 (44.9%)
Moderate, *n* (%)	140 (42.3%)	65 (41.7%)	59 (42.8%)
Severe, *n* (%)	8 (2.4%)	8 (5.1%)	0 (0%)
Treatment discontinuation			
Discontinued, *n* (%)	56 (16.9%)	28 (17.9%)	18 (13.0%)
Patient satisfaction			
High satisfaction, *n* (%)	153 (46.2%)	48 (30.8%)	84 (60.9%)
Moderate satisfaction, *n* (%)	135 (40.8%)	77 (49.4%)	46 (33.3%)
Low satisfaction, *n* (%)	43 (13.0%)	31 (19.9%)	8 (5.8%)

GI, gastrointestinal. Chi-square $p < 0.0001$ for patient satisfaction by glycemic group. Chi-square $p = 0.42$ for GI events by glycemic group (not significant)

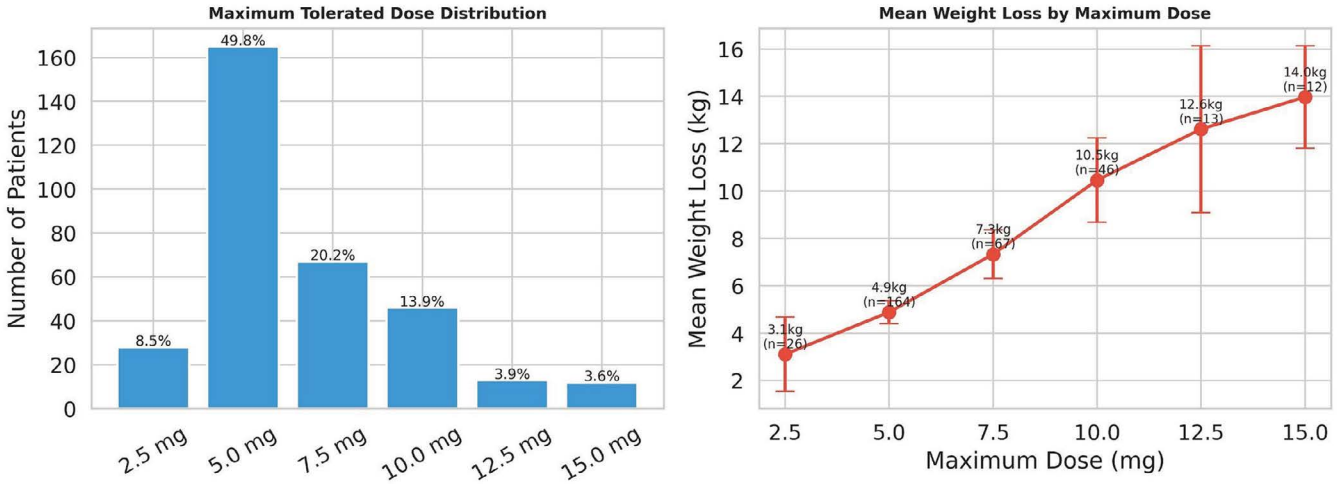

Fig. 3: Tirzepatide dosing patterns. (Left) Maximum tolerated dose distribution across the cohort ($N = 331$); the majority of patients reached 5 mg as their maximum tolerated dose (49.8%). (Right) Mean weight loss (kg) by maximum tolerated dose, demonstrating a clear dose-response relationship (Spearman $\rho = 0.47$, $p < 0.0001$). Error bars represent standard deviation

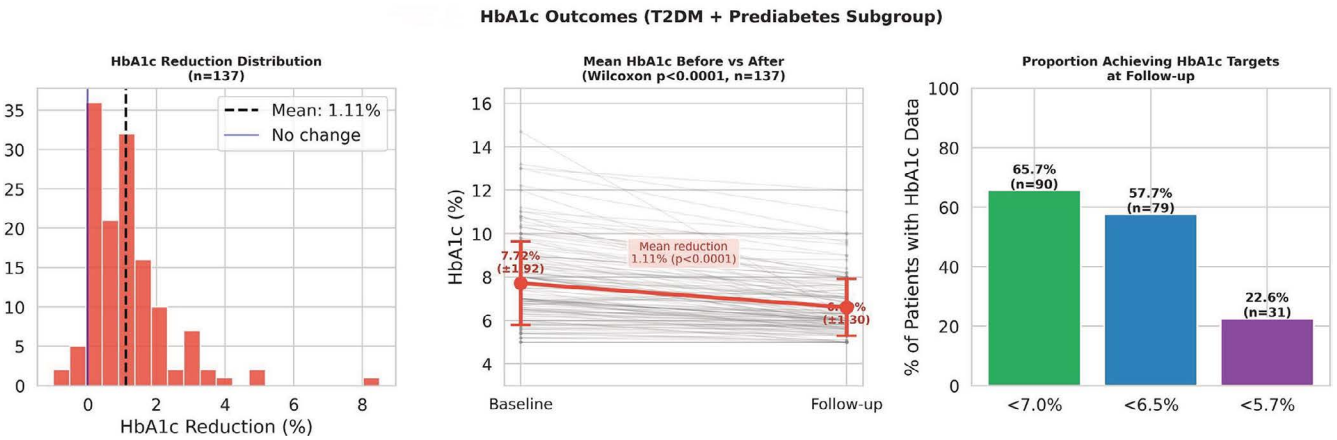

Fig. 4: HbA1c outcomes in patients with T2DM and prediabetes ($n = 137$)

Discussion

This large multicenter real-world study demonstrates that tirzepatide delivers statistically significant and clinically meaningful weight reduction and glycemic improvement in Indian adults with T2DM and/or obesity in routine clinical practice. The mean weight loss of 6.67 ± 5.03 kg (6.76% body weight; Wilcoxon $p < 0.0001$) at a median follow-up of approximately 10 weeks represents substantial early metabolic

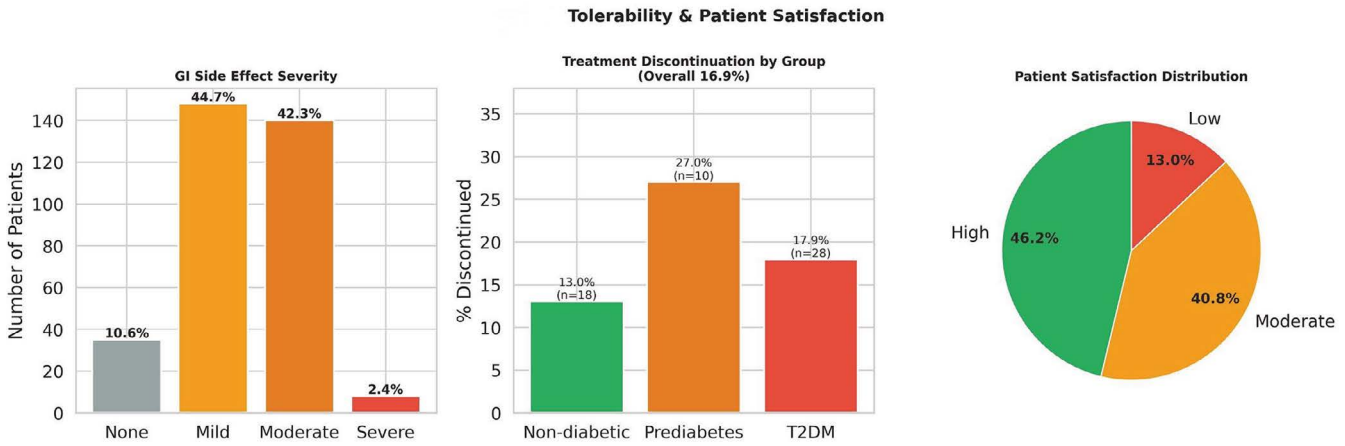

Fig. 5: Tolerability and patient satisfaction. (Left) GI side effect severity distribution across the cohort (N = 331); 89.4% reported GI events, predominantly mild to moderate. (Center) Treatment discontinuation by glycemic group (overall 16.9%); prediabetes had the highest rate (27.0%). (Right) Patient satisfaction distribution; 46.2% reported high satisfaction, driven by nondiabetic patients (60.9 vs 30.8% T2DM; Chi-square $p < 0.0001$)

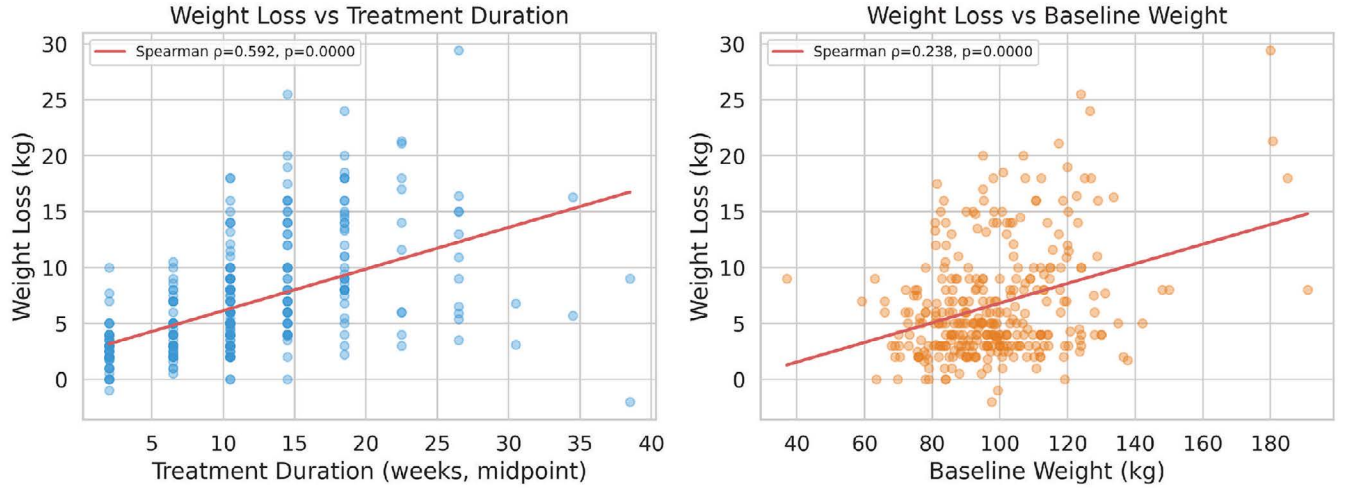

Fig. 6: Predictors of weight loss. (Left) Scatter plot of weight loss vs treatment duration (weeks, midpoint), demonstrating a significant positive correlation (Spearman $\rho = 0.592$, $p < 0.0001$). (Right) Scatter plot of weight loss vs baseline weight (kg), showing a weaker but significant positive correlation (Spearman $\rho = 0.238$, $p < 0.0001$). Red lines represent linear regression fits

benefit, particularly given the heterogeneous treatment durations and absence of the structured dose-escalation support typical of clinical trials.

The weight and glycemic outcomes are consistent with the early-phase kinetics of tirzepatide as characterized in global trials. In SURPASS-1, tirzepatide monotherapy achieved HbA1c reductions of 1.87–2.07% and weight loss of 7.0–9.5 kg over 40 weeks.[14] Our mean weight loss of 6.67 kg and HbA1c reduction of 1.11% at approximately 10 weeks of median follow-up align with the expected early-phase trajectory. The SURMOUNT-CN trial in Chinese adults demonstrated weight reductions of 13.6–17.5% at 52 weeks[18]; our 6.76% at approximately 10 weeks is consistent with this progressive weight-loss curve.

The clear dose–response relationship (Spearman $\rho = 0.47$, $p < 0.0001$) observed in this real-world cohort mirrors the dose-dependent superiority reported across the SURPASS program and confirmed in network meta-analyses of 23,622 participants from 28 RCTs.[19] The progressive increase in weight loss from 3.1 kg at 2.5 mg maintenance to 14.0 kg at 15 mg, with 100% of patients on 15 mg achieving ≥10% weight loss, reinforces the importance of adequate dose titration to maximize therapeutic benefit in clinical practice.

The differential weight loss across glycemic groups, with nondiabetic patients losing significantly more weight than T2DM patients (7.9 vs 5.4 kg; Mann–Whitney U = 7,760, p = 0.0001), is consistent with global data and mechanistically plausible: concurrent OAD/insulin use and hyperinsulinism in T2DM may attenuate net weight loss. This mirrors the SURMOUNT-2 trial (12.8–14.7% in T2DM + obesity) vs SURMOUNT-1 (15.0–20.9% in nondiabetic obesity).[16,17]

The HbA1c reduction of 1.43 ± 1.29% in the T2DM subgroup, with 65.7% achieving <7.0%, is particularly noteworthy in the Indian context. The dual GIP/GLP-1 mechanism of tirzepatide, simultaneously improving β-cell secretory function, insulin sensitivity, and glucagon suppression,[21] uniquely addresses the Indian T2DM phenotype characterized by early β-cell exhaustion and high peripheral insulin resistance.

The GI adverse event rate of 89.4% is higher than phase 3 trial rates (12–26%)[14,15] but consistent with global real-world GLP-1 RA observations, where self-reporting, variable dietary counseling, and absence of structured escalation support contribute to higher reported rates.[22-24] GI event rates did not differ significantly across glycemic groups (Chi-square p = 0.42). The 16.9% discontinuation

rate compares favorably with global real-world GLP-1 RA discontinuation rates of 20–50% within 1 year.[22] High patient satisfaction in 46.2%, driven primarily by nondiabetic patients, reinforces tirzepatide's patient acceptability profile.

Key limitations include the retrospective design, variable follow-up duration, absence of a comparator group, limited HbA1c data coverage (41.4%), and absence of body composition measurements. No dietary or lifestyle modification data were collected. Nonetheless, this study represents the largest real-world Indian multicenter experience with tirzepatide, drawn from a heterogeneous clinical population that reflects the diversity of Indian diabetes and obesity practice. Our findings are further contextualized by recently published Indian real-world studies reporting glycemic and weight benefits with injectable semaglutide and tirzepatide in Indian adults with obesity and T2DM, respectively[25,26] and by global real-world comparative data affirming tirzepatide's superiority over semaglutide on HbA1c and weight outcomes.[27,28]

Conclusion

Obesity and T2DM constitute a rapidly escalating health burden in India, compounded by the South Asian metabolic phenotype characterized by visceral adiposity, high insulin resistance, and early-onset dysglycemia at lower BMI thresholds than in Western populations.

Tirzepatide demonstrates clinically meaningful early weight reduction and glycemic improvement in Indian adults with T2DM and/or obesity in routine clinical practice, with a clear dose–response relationship (Spearman $\rho = 0.47$, $p < 0.0001$) and an acceptable tolerability profile. Nondiabetic patients achieved greater weight loss (7.9 vs 5.4 kg; $p = 0.0001$), while T2DM patients demonstrated significant HbA1c improvements, with 65.7% achieving the <7.0% target.

As the first large real-world Indian multicenter study on tirzepatide, these findings provide valuable clinical evidence supporting its effectiveness in routine Indian practice. Prospective studies with longer follow-up, body composition analysis, and cardiometabolic outcome data are warranted to fully characterize the durability and pleiotropic benefits of this therapy in the Indian phenotype.

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Orcid

Rikin Shah https://orcid.org/0009-0000-1353-6224
Sanjeev Phatak https://orcid.org/0000-0002-8270-812X
Mayur Agrawal https://orcid.org/0000-0002-7172-3690
Chirag Aghara https://orcid.org/0009-0001-3302-9585
Rucha Mehta https://orcid.org/0000-0003-3264-7987
Dhruvi Hasnani https://orcid.org/0000-0003-0535-4441
Vipul Chavda https://orcid.org/0000-0001-6071-8282

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