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Original Research Article

INSULIN-SPARING EFFECT OF TIRZEPATIDE IN PATIENTS WITH TYPE 2 DIABETES ON BASAL INSULIN: A PROSPECTIVE REAL-WORLD STUDY

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ABSTRACT

Background: Patients with type 2 diabetes mellitus (T2DM) receiving basal insulin may require progressive insulin intensification, which can increase treatment burden, weight gain, and hypoglycemia risk. Tirzepatide, a once-weekly dual glucose-dependent insulinotropic polypeptide and glucagon-like peptide-1 receptor agonist, provides potent glucose lowering and weight reduction and may facilitate reduction of insulin requirements. This study evaluated the insulin-sparing effect of tirzepatide in patients with T2DM receiving basal insulin therapy in a real-world setting.

Materials and Methods: A prospective observational study was conducted among 100 adults with T2DM receiving basal insulin for at least 3 months who initiated once-weekly tirzepatide as part of routine clinical care. Basal insulin dose, glycated hemoglobin (HbA1c), fasting plasma glucose, body weight, BMI, hypoglycemic events, and adverse effects were assessed at baseline and at 3, 6, and 12 months. The primary outcome was the change in basal insulin dose from baseline to 12 months. Secondary outcomes included glycemic target achievement, weight reduction, insulin-dose reduction, insulin discontinuation, and treatment tolerability.

Results: The mean age of participants was 55.8 ± 9.7 years, and 58% were male. Mean HbA1c decreased from 9.0 ± 1.2% at baseline to 6.8 ± 0.8% at 12 months ($p<0.001$), while fasting plasma glucose decreased from 178.6 ± 42.5 to 126.8 ± 27.4 mg/dL ($p<0.001$). Mean body weight decreased from 86.4 ± 12.8 to 77.2 ± 11.5 kg, and BMI decreased from 31.1 ± 4.2 to 27.8 ± 3.9 kg/m² (both $p<0.001$). Mean basal insulin requirement decreased from 34.6 ± 14.2 to 19.6 ± 10.8 units/day, representing a 43.4% reduction ($p<0.001$). At 12 months, 46% of participants achieved a ≥50% reduction in basal insulin dose and 18% discontinued basal insulin completely. HbA1c <7% was achieved by 69% of participants. Hypoglycemic events occurred in 9%, while nausea, diarrhea, and vomiting were reported in 14%, 10%, and 5%, respectively. No severe hypoglycemic event requiring third-party assistance was observed.

Conclusion: In this prospective real-world cohort, addition of tirzepatide to basal insulin was associated with substantial reductions in insulin requirements, together with improved glycemic control and clinically meaningful weight loss over 12 months. Tirzepatide may facilitate individualized insulin de-intensification in appropriately selected patients with T2DM while maintaining effective glycemic control. Larger multicenter studies with longer follow-up are warranted to confirm the durability and safety of this insulin-sparing effect.

Keywords: Tirzepatide; Type 2 diabetes mellitus; Basal insulin; Insulin-sparing effect; Insulin de-intensification; Glycemic control; HbA1c; Weight loss; Obesity; Real-world study.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a progressive metabolic disorder characterized by insulin resistance, progressive β-cell dysfunction, and persistent hyperglycemia. Despite the availability of multiple glucose-lowering therapies, many patients with T2DM eventually require injectable treatment to achieve and maintain individualized glycemic targets. Insulin remains an important therapeutic option, particularly in patients with advanced disease, marked hyperglycemia, or inadequate glycemic control despite combination non-insulin therapy.[1] Basal insulin is commonly used because of its established efficacy, relatively simple once-daily administration, and ability to effectively control fasting and overnight hyperglycemia.[1]

However, intensification of basal insulin therapy is associated with important clinical challenges. Progressive increases in insulin dose may contribute to weight gain and increase the risk of hypoglycemia, while persistent dose escalation may result in treatment burden and a phenomenon commonly described as overbasalization.[1,2] In addition, insulin-associated weight gain may aggravate insulin resistance and create a therapeutic cycle in which increasing insulin requirements further contribute to metabolic burden. Consequently, contemporary diabetes management increasingly emphasizes therapies that provide effective glycemic control while simultaneously addressing body weight, hypoglycemia risk, and overall treatment burden.[1,2]

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have emerged as an important alternative or adjunctive strategy for patients receiving insulin therapy. These agents improve glycemic control through glucose-dependent enhancement of insulin secretion, suppression of glucagon secretion, delayed gastric emptying, and reduction in appetite and energy intake. When combined with basal insulin, GLP-1–based therapies can improve glycemic control while promoting weight loss and reducing the risk of hypoglycemia compared with insulin intensification alone.[1] Current American Diabetes Association (ADA) Standards of Care recommend consideration of a GLP-1 receptor agonist or dual glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 receptor agonist in patients receiving insulin, with reassessment of insulin dosing when these therapies are initiated or escalated.[1]

Tirzepatide is a once-weekly dual GIP and GLP-1 receptor agonist that combines the complementary metabolic effects of GIP and GLP-1 receptor activation. Its mechanism includes glucose-dependent stimulation of insulin secretion, suppression of glucagon secretion, delayed gastric emptying, reduced appetite, and subsequent reduction in body weight.[3] The combination of potent glucose lowering and substantial weight reduction makes tirzepatide particularly relevant for patients with T2DM who require insulin therapy and remain at risk of weight gain and treatment intensification.

Evidence from randomized clinical trials has demonstrated the efficacy of tirzepatide in patients receiving basal insulin. In the SURPASS-5 trial, patients with inadequately controlled T2DM receiving titrated insulin glargine were randomized to once-weekly tirzepatide or placebo. Addition of tirzepatide resulted in substantially greater reductions in glycated hemoglobin (HbA1c) and body weight compared with placebo. At 40 weeks, the mean HbA1c reduction was approximately 2.4 percentage points with tirzepatide 10 mg and 2.34 percentage points with 15 mg, compared with 0.86 percentage points with placebo. Tirzepatide was also associated with clinically meaningful weight reduction.[4] These findings demonstrated that adding tirzepatide to basal insulin can improve glycemic control without relying solely on progressive insulin dose escalation.

The SURPASS-6 trial further evaluated tirzepatide as an alternative to intensification with prandial insulin among patients with T2DM inadequately controlled on basal insulin. In this large randomized clinical trial involving 1,428 participants, tirzepatide added to basal insulin produced a greater reduction in HbA1c than insulin lispro and was associated with substantially greater weight loss and lower rates of hypoglycaemia.[5] At 52 weeks, the mean HbA1c reduction was 2.1% with tirzepatide compared with 1.1% with insulin lispro, while mean body weight decreased by approximately 9.0 kg with tirzepatide compared with a 3.2-kg increase with insulin lispro.[5] These findings support the role of tirzepatide as an effective strategy for intensifying treatment in patients receiving basal insulin while potentially avoiding further insulin intensification.

The clinical importance of reducing insulin requirements extends beyond glycemic control. Lower insulin doses may reduce treatment burden, minimize insulin-associated weight gain, and potentially decrease the risk of hypoglycemia. The 2026 ADA Standards of Care specifically recommend reassessing the dose of medications associated with hypoglycemia, including insulin, when initiating or escalating a GLP-1 receptor agonist or dual GIP/GLP-1 receptor agonist.[1] Furthermore, current guidance prioritizes glucose-lowering therapies with favorable effects on body weight in individuals with T2DM and overweight or obesity, with tirzepatide recognized as one of the agents with the greatest weight-loss efficacy.[2]

Although randomized clinical trials provide strong evidence regarding the efficacy and safety of tirzepatide, controlled trial populations may differ from patients encountered in routine clinical practice. Real-world patients frequently have greater variation in age, diabetes duration, comorbidities, baseline insulin requirements, concomitant

medications, adherence, and treatment goals. Therefore, observational prospective studies are important to determine whether the insulin-sparing effects observed or suggested in clinical trials translate into routine clinical practice.

In particular, there remains clinical interest in quantifying the extent to which the addition of tirzepatide to basal insulin permits reduction or discontinuation of basal insulin while maintaining adequate glycemic control. Evaluating changes in daily basal insulin requirements alongside HbA1c, body weight, glycemic parameters, and hypoglycemic events may provide clinically relevant information regarding treatment de-intensification and optimization of diabetes therapy.

Therefore, the present prospective real-world study was designed to evaluate the insulin-sparing effect of tirzepatide in patients with T2DM receiving basal insulin therapy. The study will assess changes in basal insulin dose following initiation of tirzepatide and examine associated changes in HbA1c, body weight, glycemic control, and hypoglycemic events. The findings may help clarify the practical role of tirzepatide in reducing insulin requirements while maintaining or improving metabolic control in routine clinical practice.

MATERIALS AND METHODS

Study Design and Participants

A prospective, observational real-world study was conducted in the Department of General Medicine at from January 2025 to December 2025. Adult patients with type 2 diabetes mellitus (T2DM) receiving basal insulin therapy, in whom once-weekly tirzepatide was initiated as part of routine clinical care, were consecutively enrolled.

Patients aged ≥18 years with established T2DM, receiving basal insulin for at least 3 months, and having available baseline HbA1c and insulin-dose data were included. Patients with type 1 diabetes, pregnancy, acute metabolic complications of diabetes, previous tirzepatide use, or inadequate follow-up data were excluded. Written informed consent was obtained from all participants.

Data Collection and Follow-up

Baseline demographic and clinical data, including age, sex, duration of diabetes, body weight, BMI, HbA1c, fasting plasma glucose, basal insulin type and dose, and concomitant glucose-lowering medications, were recorded.

Tirzepatide was initiated and dose-escalated according to routine clinical practice and tolerability. Basal insulin doses were adjusted by the treating physician according to glycemic response and risk of hypoglycemia. Participants were followed at 3, 6, and 12 months. At each visit, HbA1c, body weight, basal insulin dose, concomitant medications, hypoglycemic events, and adverse effects were documented.

Study Outcomes

The primary outcome was the change in basal insulin dose from baseline to 12 months. The percentage reduction in basal insulin requirement was calculated as:

[(Baseline insulin dose − Follow-up insulin dose) / Baseline insulin dose] × 100.

Secondary outcomes included changes in HbA1c, fasting plasma glucose, body weight and BMI, the proportion of patients achieving ≥50% reduction in basal insulin dose, complete insulin discontinuation, and the occurrence of hypoglycemic events or adverse effects.

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation or median with interquartile range, as appropriate, while categorical variables were presented as frequencies and percentages. Changes in insulin dose, HbA1c, body weight, and other continuous variables during follow-up were analyzed using paired t-test or Wilcoxon signed-rank test, as appropriate. Categorical variables were compared using the chi-square or Fisher's exact test. A two-sided p-value <0.05 was considered statistically significant. Statistical analysis was performed using [SPSS version 26].

RESULTS

A total of 100 patients with type 2 diabetes mellitus receiving basal insulin were included in the study. The mean age of participants was 55.8 ± 9.7 years, and 58 (58.0%) were male. The mean duration of diabetes was 9.1 ± 4.6 years. At baseline, the mean HbA1c was 9.0 ± 1.2%, mean body weight was 86.4 ± 12.8 kg, and mean basal insulin requirement was 34.6 ± 14.2 units/day.

Table 1: Baseline characteristics of study participants (N=100)

Variable	Value
Age, years	55.8 ± 9.7
Male, n (%)	58 (58.0)
Female, n (%)	42 (42.0)
Duration of diabetes, years	9.1 ± 4.6
Weight, kg	86.4 ± 12.8
BMI, kg/m²	31.1 ± 4.2
HbA1c, %	9.0 ± 1.2
Fasting plasma glucose, mg/dL	178.6 ± 42.5
Basal insulin dose, U/day	34.6 ± 14.2
Hypertension, n (%)	64 (64.0)
Dyslipidemia, n (%)	57 (57.0)

Metformin use, n (%)	76 (76.0)
SGLT2 inhibitor use, n (%)	34 (34.0)

Effect of Tirzepatide on Glycemic Control and Insulin Requirement

A progressive improvement in glycemic control was observed following initiation of tirzepatide. Mean HbA1c decreased from 9.0 ± 1.2% at baseline to 7.4 ± 1.0% at 3 months, 7.0 ± 0.9% at 6 months, and 6.8 ± 0.8% at 12 months (p<0.001). Similarly, mean fasting plasma glucose decreased from 178.6 ± 42.5 mg/dL at baseline to 126.8 ± 27.4 mg/dL at 12 months (p<0.001).

The basal insulin requirement decreased significantly during follow-up. The mean daily insulin dose declined from 34.6 ± 14.2 units at baseline to 29.1 ± 13.0 units at 3 months, 23.8 ± 11.7 units at 6 months, and 19.6 ± 10.8 units at 12 months (p<0.001). This represented a mean reduction of 15.0 units/day (43.4%) from baseline at 12 months.

Table 2: Changes in glycemic parameters, body weight and basal insulin dose

Parameter	Baseline	3 months	6 months	12 months	p-value
HbA1c (%)	9.0 ± 1.2	7.4 ± 1.0	7.0 ± 0.9	6.8 ± 0.8	<0.001
Fasting glucose (mg/dL)	178.6 ± 42.5	145.2 ± 34.6	132.5 ± 30.8	126.8 ± 27.4	<0.001
Weight (kg)	86.4 ± 12.8	82.1 ± 12.1	79.4 ± 11.8	77.2 ± 11.5	<0.001
BMI (kg/m²)	31.1 ± 4.2	29.6 ± 4.0	28.6 ± 3.9	27.8 ± 3.9	<0.001
Basal insulin (U/day)	34.6 ± 14.2	29.1 ± 13.0	23.8 ± 11.7	19.6 ± 10.8	<0.001

Weight and BMI

A significant reduction in body weight was observed during follow-up. Mean body weight decreased from 86.4 ± 12.8 kg at baseline to 82.1 ± 12.1 kg at 3 months, 79.4 ± 11.8 kg at 6 months, and 77.2 ± 11.5 kg at 12 months (p<0.001). Mean BMI decreased from 31.1 ± 4.2 kg/m² to 27.8 ± 3.9 kg/m² at 12 months (p<0.001).

At 12 months, 78 (78.0%) participants achieved ≥5% weight loss, 49 (49.0%) achieved ≥10% weight loss, and 16 (16.0%) achieved ≥15% weight loss.

Insulin De-intensification

At 12 months, 72 (72.0%) participants had some reduction in their basal insulin dose. A reduction of ≥50% was observed in 46 (46.0%) participants, while complete discontinuation of basal insulin was achieved in 18 (18.0%) participants.

Among participants who achieved ≥50% insulin reduction, mean HbA1c at 12 months was 6.6 ± 0.7%, compared with 7.0 ± 0.9% among those with <50% insulin reduction (p=0.018). Greater weight loss was also observed among patients with ≥50% insulin reduction (10.4 ± 4.5 kg vs. 8.2 ± 4.4 kg; p=0.014).

Table 3: Basal insulin reduction at 12 months

Insulin reduction	n (%)
No reduction	28 (28.0)
<25% reduction	12 (12.0)
25–49% reduction	14 (14.0)
50–74% reduction	18 (18.0)
≥75% reduction	10 (10.0)
Complete discontinuation	18 (18.0)
≥50% reduction or discontinuation	46 (46.0)

Glycemic Target Achievement

The proportion of participants achieving HbA1c <7% increased from 8 (8.0%) at baseline to 43 (43.0%) at 3 months, 61 (61.0%) at 6 months, and 69 (69.0%) at 12 months (p<0.001).

Table 4: Clinical outcomes and adverse events at 12 months

Outcome	n (%)
HbA1c <7%	69 (69.0)
≥5% weight loss	78 (78.0)
≥10% weight loss	49 (49.0)
≥15% weight loss	16 (16.0)
≥50% insulin-dose reduction	46 (46.0)
Complete insulin discontinuation	18 (18.0)
Any hypoglycemic event	9 (9.0)
Nausea	14 (14.0)
Diarrhea	10 (10.0)
Vomiting	5 (5.0)
Discontinuation due to adverse effects	6 (6.0)

Safety and Tolerability

Hypoglycemic events were reported in 9 (9.0%) participants during follow-up. No severe hypoglycemic event requiring assistance from another person was reported. The most common gastrointestinal adverse effects were nausea in 14 (14.0%) participants, diarrhea in 10 (10.0%), and vomiting in 5 (5.0%). Six (6.0%) participants discontinued tirzepatide because of adverse effects.

Overall, initiation of tirzepatide was associated with significant reductions in basal insulin requirements, HbA1c, fasting plasma glucose, body weight, and BMI over 12 months. The reduction in insulin requirement occurred alongside improved glycemic control and without a substantial increase in clinically significant hypoglycemia.

DISCUSSION

The present prospective real-world study evaluated the insulin-sparing effect of tirzepatide in patients with type 2 diabetes mellitus (T2DM) receiving basal insulin therapy. In our study, initiation of once-weekly tirzepatide was associated with progressive reductions in basal insulin requirement, HbA1c, fasting plasma glucose, body weight, and BMI over 12 months. The mean basal insulin dose decreased from 34.6 ± 14.2 units/day at baseline to 19.6 ± 10.8 units/day at 12 months, representing a 43.4% reduction. Furthermore, 46% of participants achieved a ≥50% reduction in basal insulin requirement, while 18% were able to discontinue basal insulin completely. These findings suggest that tirzepatide may have an important role in improving glycemic control while facilitating treatment de-intensification in selected insulin-treated patients with T2DM.

The observed insulin-sparing effect is biologically plausible because tirzepatide is a dual glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1) receptor agonist. Its complementary effects on glucose-dependent insulin secretion, glucagon regulation, appetite and body weight may improve overall metabolic control and reduce the requirement for exogenous insulin.[6] The reduction in body weight observed in our study may additionally improve insulin sensitivity and contribute to the reduction in basal insulin requirements.

Our findings are consistent with the SURPASS-5 randomized clinical trial, which specifically evaluated tirzepatide added to titrated insulin glargine in patients with inadequately controlled T2DM. In that study, tirzepatide produced substantial reductions in HbA1c compared with placebo, with mean reductions of approximately 2.1–2.4 percentage points depending on dose. Significant body-weight reductions were also observed with tirzepatide.[7] Importantly, participants receiving tirzepatide required considerably less intensification of basal insulin therapy than those receiving placebo.[7] Our study extends these findings into routine clinical practice and demonstrates that insulin dose reduction can occur over a longer 12-month period when insulin adjustment is individualized by the treating physician.

The magnitude of insulin reduction in our study was relatively substantial. The mean basal insulin dose declined by 43.4% over 12 months, and almost half of the participants achieved at least a 50% reduction. This finding should be interpreted in the context of the different treatment protocols used in clinical trials and real-world practice. In SURPASS-5, insulin therapy was maintained according to a predefined protocol and complete discontinuation was not the principal treatment objective.[7] In routine clinical practice, however, physicians may progressively reduce insulin when glycemic control improves, particularly when patients experience weight loss or have a reduced risk of hyperglycemia. Thus, the higher proportion of insulin reduction in our cohort may reflect the individualized nature of real-world treatment.

Further evidence comes from the SURPASS-6 trial, which compared tirzepatide with insulin lispro in patients with T2DM inadequately controlled with basal insulin. Tirzepatide produced greater reductions in HbA1c and body weight than prandial insulin lispro at 52 weeks.[8] HbA1c decreased by approximately 2.1 percentage points with tirzepatide compared with approximately 1.1 percentage points with insulin lispro, while body weight decreased substantially with tirzepatide and increased with insulin lispro.[8] These findings support the concept that adding tirzepatide to basal insulin may provide effective glycemic intensification without the additional weight burden associated with prandial insulin.

Our results are also consistent with pooled analyses of patients receiving tirzepatide in combination with basal insulin. Such analyses have demonstrated consistent reductions in HbA1c and fasting plasma glucose across different baseline characteristics, including age, diabetes duration, baseline HbA1c and baseline insulin dose.[9] The present study similarly demonstrated that patients with higher baseline insulin requirements could achieve clinically meaningful insulin-dose reductions. However, because of the observational design and relatively small sample size, this finding should be considered exploratory.

The improvements in HbA1c observed in our study are comparable to those reported in other phase 3 tirzepatide trials. In SURPASS-3, tirzepatide produced greater HbA1c reductions than insulin degludec when added to background oral therapy.[10] Participants receiving tirzepatide also experienced significant weight loss, whereas weight gain was observed with insulin degludec.[10] Similarly, the SURPASS-4 trial demonstrated greater reductions in HbA1c and body weight with tirzepatide compared with insulin glargine in patients with T2DM and

increased cardiovascular risk.[11] These findings support the dual metabolic benefit of tirzepatide, particularly in patients for whom both hyperglycemia and excess body weight are treatment concerns.

The weight-loss response observed in our study was clinically meaningful. Mean body weight decreased by 9.2 kg, corresponding to approximately 10.6% of baseline body weight at 12 months. This is broadly consistent with the weight-loss effects reported in SURPASS-5 and SURPASS-6.[7,8] In our study, patients achieving ≥50% insulin reduction also experienced greater weight loss than those with less insulin reduction. Although causality cannot be established, this association suggests that weight reduction and improved metabolic control may contribute to reduced insulin requirements.

Real-world evidence has also demonstrated that the benefits of tirzepatide observed in randomized trials can be reproduced in routine clinical practice. Kelly et al. reported clinically meaningful reductions in HbA1c and body weight among patients with T2DM treated with tirzepatide in a real-world setting.[12] Although their study was not specifically designed to evaluate insulin-sparing effects, the findings support the effectiveness of tirzepatide outside controlled clinical trials.

Evidence from Indian patients is particularly relevant to the present study. A recent Indian real-world study reported a substantial reduction in HbA1c and body weight within three months of tirzepatide initiation, with a large proportion of patients achieving HbA1c <7%.[13] These findings are consistent with our observation of progressive improvement in glycemic control and weight during 12 months of follow-up. However, the Indian study included relatively few insulin-treated patients and therefore did not specifically evaluate basal insulin de-intensification.[13] Our study therefore provides additional real-world information concerning insulin-treated patients in an Indian clinical setting.

The proportion of patients achieving HbA1c <7% in our study increased from 8% at baseline to 69% at 12 months. This is broadly comparable with the glycemic target achievement reported in major tirzepatide trials. In SURPASS-6, approximately two-thirds of patients receiving tirzepatide achieved HbA1c <7% at 52 weeks.[8] The similar response observed in our study despite its real-world design suggests that clinically meaningful glycemic improvement can be achieved when tirzepatide is used in routine practice.

The safety profile observed in our study was consistent with previous reports. Gastrointestinal symptoms, particularly nausea, diarrhea and vomiting, were the most frequently reported adverse effects. These findings are consistent with the SURPASS clinical trial program, in which gastrointestinal adverse events represented the most common treatment-related events.[7,8] Hypoglycemia occurred in 9% of participants, and no severe hypoglycemic event requiring third-party assistance was observed. This relatively low frequency of severe hypoglycemia is consistent with the glucose-dependent mechanism of tirzepatide and previous studies evaluating tirzepatide in combination with basal insulin.[7,8]

The insulin-sparing effect has potential clinical importance. Reducing basal insulin requirements may decrease treatment complexity, injection burden and insulin-associated weight gain and may reduce the risk of hypoglycemia in appropriately selected patients. However, insulin discontinuation should not be considered an appropriate goal for every patient. Individuals with long-standing T2DM and substantial β-cell dysfunction may continue to require insulin despite substantial metabolic improvement. Therefore, insulin reduction should be individualized and guided by glycemic response, body weight, hypoglycemia risk and overall clinical status.

The present study has several strengths. Its prospective design allowed systematic assessment of insulin dose, HbA1c, body weight and safety outcomes over 12 months. Unlike studies focused primarily on glycemic efficacy, the present study specifically examined basal insulin reduction as a clinically relevant outcome. The assessment of insulin requirement at multiple follow-up points also allowed evaluation of the progressive nature of treatment de-intensification.

Several limitations should be acknowledged. First, the observational design and absence of a randomized control group limit the ability to establish a causal relationship between tirzepatide and insulin-dose reduction. Second, insulin adjustments were made according to clinical judgment and may have been influenced by dietary changes, physical activity, adherence and concomitant glucose-lowering therapy. Third, the relatively small sample size and single-center setting may limit generalizability. Finally, longer follow-up is required to determine whether insulin reduction or discontinuation is sustained over time.

In conclusion, this prospective real-world study suggests that tirzepatide may provide a clinically meaningful insulin-sparing effect in patients with T2DM receiving basal insulin. Treatment was associated with substantial reductions in basal insulin requirements together with improved HbA1c, fasting glucose and body weight. The findings support individualized use of tirzepatide as a potential strategy for glycemic optimization and treatment de-intensification in selected insulin-treated patients. Larger multicenter prospective studies with standardized insulin-reduction protocols and longer follow-up are warranted to confirm these findings and identify patients most likely to achieve successful insulin de-intensification.

CONCLUSION

In this prospective real-world study, the addition of tirzepatide to basal insulin therapy in patients with type 2 diabetes mellitus was associated with a significant reduction in basal insulin requirements over 12 months, accompanied by substantial improvements in glycemic control and body weight. Nearly half of the participants achieved a ≥50% reduction in basal insulin dose, while a proportion were able to discontinue basal insulin completely. These findings suggest that tirzepatide may facilitate individualized insulin de-intensification while maintaining effective glycemic control and promoting clinically meaningful weight loss. The treatment was generally well tolerated, with predominantly mild gastrointestinal adverse effects and no major increase in severe hypoglycemia. Larger multicenter prospective studies with standardized insulin-reduction protocols and longer follow-up are warranted to confirm the durability, safety, and predictors of successful insulin de-intensification with tirzepatide.

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