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A Retrospective Analysis of Lobeglitazone as an Add-On to Existing Glucose-Lowering Therapy in Indian Adults with Suboptimally Controlled Type 2 Diabetes for Its Clinical Effectiveness: A Real-World Clinical Experience

ABSTRACT

Objective: The objective of the current study was to evaluate the safety and effectiveness of lobeglitazone as an add-on therapy in suboptimally controlled Indian type 2 diabetes (T2D) patients in a real-world clinical setup.

Materials and methods: The study was conducted in suboptimally controlled T2D patients while being treated with lobeglitazone once daily (0.5 mg) as an add-on to existing glucose-lowering agents in various clinics in eastern India.

Results: The patients' average body weight was 80.78 ± 9.36 kg, with a body mass index (BMI) of 30.86 ± 4.16 kg/m^2. The addition of lobeglitazone 0.5 mg to existing therapy over 12 weeks resulted in a statistically significant HbA1c reduction (–1.1 ± 0.72, $p < 0.005$). Among all 4 groups, similar glycemic declines were observed with no major intergroup variation ($p = 0.074$). Also, there was a statistically significant mean drop in both postprandial plasma glucose (–71.47 ± 26.73, $p < 0.005$) and fasting plasma glucose (FPG) (–47.11 ± 21.45, $p < 0.005$). It was found that a patient's BMI was significantly linked to their likelihood of meeting their recommended target HbA1c (57.3% in BMI 25–30 vs. 34% in BMI > 30, $p = 0.0233$) and target FPG (59% in BMI 25–30 vs. 45.3% in BMI > 30, $p = 0.0112$). Among the total population, 35 (9.61%) patients reported hypoglycemia, and no one required medical assistance.

Conclusions: In suboptimally controlled diabetes patients, in combination with one or more commonly prescribed antidiabetic drugs, lobeglitazone significantly improved the glycemic and non-glycemic measures and was well tolerated. (Clin Diabetol 2024; 13, 5: 274–281)

Keywords: lobeglitazone, thiazolidinediones, type 2 diabetes

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Introduction

Diabetes, an exemplary chronic disease, has reached epidemic proportions not only in India but also worldwide. It is estimated that the global burden of type 2 diabetes (T2D) is expected to increase to 592 million by 2035 [1]. There were over 72.9 million cases of diabetes in India in 2017 [2]. There is evidence of an epidemiological transition, with diabetes prevalence being higher in urban regions with lower socioeconomic categories in states with higher levels of economic development. There is sufficient evidence of an "Asian phenotype" in diabetes [3]. Asians have a 2–4-fold higher risk of T2D than white Europeans, independent of weight, and develop diabetes 5–10 years earlier than them [4].

Metformin is considered the most widely used glucose lowering agent worldwide and for any individual diagnosed with type 2 diabetes, the American Diabetes Association (ADA) recommends metformin treatment and that glycated hemoglobin (HbA1c) be maintained at ≤ 7% [5]. ADA also recommends that when patients have a high baseline HbA1c (≥ 9.0%), a combination of 2 non-insulin agents should be used to achieve the target HbA1c [6]. It has been hypothesized that long-term glycemic effectiveness or 'durability' is maintained when metformin combines with an agent from another class with a different mechanism of action and therefore also helps preserve β-cell function [7]. The major classes of oral antidiabetic medications include biguanides, thiazolidinedione (TZD), sulfonylureas, sodium-glucose cotransporter (SGLT2) inhibitors, α-glucosidase inhibitors, dipeptidyl peptidase 4 (DPP-4) inhibitors, and meglitinide.

There are several classes of drugs available in the Indian market, even though the burden of suboptimally controlled diabetes in India is increasing day by day. Lobeglitazone was recently approved as a glucose-lowering agent in India by the Indian drug regulator, the Drug Controller General of India. Lobeglitazone belongs to the group of thiazolidinediones (TZDs), which act as agonists of peroxisome proliferator-activated receptor-γ [5]. Lobeglitazone is a selective and potent agonist of PPAR-γ and is an insulin sensitizer acting on intracellular metabolic pathways to enhance insulin action and increase insulin sensitivity in critical tissues [5–7]. Lobeglitazone activates PPAR-γ, which leads to a decrease in insulin resistance, facilitating differentiation of mesenchymal stem cells into adipocytes, enhancing lipogenesis in peripheral adipocytes. Also, there is a decrease in hepatic and peripheral triglyceride, a decrease in visceral adipocytes, and an increase in adiponectin [5]. This markedly ameliorates insulin resistance, and the metabolic syndrome and decreases insulin requirements [8].

There have been few international studies demonstrating that lobeglitazone reduces blood sugar levels, lowers HbA1c levels, and improves lipid and liver profiles by activating PPAR-γ and promoting the binding of insulin to fat cells [9, 10]. Lobeglitazone has been designed by modifying the rosiglitazone structure with a substituted pyrimidine; it has a p-methoxyphenoxy group at the 4-position of the pyrimidine moiety [9, 11]. It has additional hydrophobic contacts with the ligand-binding pocket that account for its enhanced affinity and the low effective dose compared to those of rosiglitazone and pioglitazone. Also, lobeglitazone displays 12-fold higher affinity to PPAR-γ than rosiglitazone and pioglitazone [11, 12].

There is a clinically unmet need for an effective antidiabetic treatment with a lower propensity to weight gain and hypoglycemic events that can ameliorate the risk of progression to major complications. Thus, we need a newer therapy with superior effectivity with simplified drug dosing that can target various stages of the disease and also demonstrate favorable safety profiles and play a role in improving patient adherence and quality of life. The objective of the current study was to evaluate the safety and effectiveness of lobeglitazone as an add-on therapy in suboptimally controlled Indian type 2 diabetes patients in a real-world clinical setting.

Materials and methods

Study design

This is a single arm observational, retrospective, multicenter study conducted on adult Indian suboptimally controlled (HbA1c > 7% within 2 weeks or at the initiation) type 2 diabetes patients aged between 18 and 70 years. Patient data were retrieved from the electronic medical records of the respective diabetic clinic, and during the analysis of the patient records the study conformed to the ethical principles of the Declaration of Helsinki. Informed consent was not obtained because of the unidentified nature of the patients' data.

Study population

Initially, 550 patients with data from at least a 12-week follow-up were selected, but at the end of screening, 364 records were eligible for final analysis. Supplementary Figure 1 illustrates the patient flow in the study. This retrospective study is a 12-week analysis.

Inclusion criteria

Men and female Indian patients with a clinical diagnosis of suboptimally controlled T2D (HbA1c > 7% within 2 weeks or at the initiation) and aged between 18 and 70 years were included in the study, and all

patients were on one or more glucose-lowering medications for at least one month before inclusion. All participants had been diagnosed with type 2 diabetes for at least one year. All included patients had all recorded study parameters recorded at periodic intervals.

Exclusion criteria

Individuals who were diagnosed with type 1 diabetes, with a cardiovascular history, or with any history of kidney disease, with any history of recurrent genitourinary tract infection, diabetic ketoacidosis, or bone fracture in the 6 months before screening generally did not opt to start with a new drug, especially a drug belonging to the thiazolidinedione (TZD) group.

Data collection/variables

Monitoring for adverse experiences, physical examinations, vital signs, body weight, and electrocardiogram (ECG) and laboratory measurements comprising routine hematology, serum chemistry, and urinalysis were performed in a NABL accredited pathology laboratory attached to the study centers.

By using standardized procedures, anthropometric measurements were recorded. For each weight measurement (baseline as week 0 and follow-up visits), the body mass index (BMI) was calculated. In OPD records for glycemic profile, blood tests at baseline, subsequently on every visit, and at the end of 12 weeks (weeks 0, 1, 3, 5, 7, 9, and 12) were examined. For all participants, self-monitoring of blood glucose (SMBG) was advised, which was further captured in each patient record. For the analysis of changes in baseline and post treatment with lobeglitazone, subjects were further divided into 4 groups as per the number of drugs they were consuming. The patients' vital parameters like body weight and blood pressure were recorded at each visit. During each follow-up visit, the occurrence of any adverse event was recorded and examined. All patients attended the respective clinic as per the visit dates mentioned on their prescription, and the process was strictly monitored. To record the socio-demographic parameters, a closed-ended semi-structured proforma form was used. The hexokinase method of glucose estimation was performed to evaluate fasting and postprandial blood glucose (Abbot Model Aclyon 300, USA auto analyzer with Pars-Azmone kit) (normal range of fasting plasma glucose 80-100 mg/dl and for postprandial 120–140 mg/dL). Ion-exchange high--performance liquid chromatography (HPLC) methods were used to determine HbA1c (Merilyzer GluQuant) (normal value < 6.5%). Serum Cr was measured via the visible absorption spectrometry enzymatic method (StatSensor) (normal result 0.7 to 1.3 mg/dL for men and 0.6 to 1.1 mg/dL for women). Total cholesterol and triglyceride (TG) levels were determined using the cholesterol dehydrogenase UV method and an enzymatic method (normal value < 200 mg/dL). High-density lipoprotein (HDL) cholesterol levels were measured via a direct assay using Cholestest N-HDL (normal value > > 60 mg/dL). Low-density lipoprotein (LDL) cholesterol levels were measured using the Friedewald formula (Normal value < 100 mg/dL).

Ethical approval

The study was exempt from ethical committee approval because it was confined to anonymized and unidentifiable data routinely collected at our Diabetes Center.

Statistical analysis

A minimum of 350 patients were needed to achieve above 80% power, assuming a standard deviation of 1% and $\alpha = 0.017$ (corrected for 3 possible comparisons) to identify a 0.4% treatment difference in HbA1c with lobeglitazone 0.5 mg.

The change in HbA1c from baseline over the treatment period (week 12) was the primary end point. Secondary endpoints were change in fasting plasma glucose FPG, post prandial plasma glucose (PPPG), body weight, systolic BP, diastolic BP, creatinine LDL-C, HDL-C and triglycerides from baseline to the end of the treatment period (week 12), percentage of patients receiving rescue therapy, and the proportion of patients achieving HbA1c less than 7.0% at week 12.

Excel sheets were used to present the data, and data were analyzed statistically using GraphPad prism (version 8.4) software. By using the mean and standard deviation, quantitative variables were summarized. The dependent sample t-test or Student's t-test was used to test the significance of differences between the mean values of 2 continuous variables. To obtain odds ratios by using a logistic regression model in group comparison, binary endpoints of the likelihood of achieving the target HbA1c level ware analyzed. By frequencies and percentages, the categorical values were described, and at an alpha level of 0.05 the chi-square test was applied.

Results

The characteristic baseline data of the participants

Baseline characteristics and demographics were illustrated in Table 1. Average mean age among participants was 58.32 ± 7.1 years, and 51.4% were male. The average body weight was 80.78 ± 9.36 kg, with a BMI of 30.86 ± 4.16 kg/m^2, indicating mainly obese or overweight patients taking the present

Table 1. Baseline Characteristics at the Initiation of Lobeglitazone as an Add-On to Existing Glucose-Lowering Therapy (N = 364)

Baseline Parameters	Observation
Age [years], mean (SD)	58.32 ± 7.1
Male, N (%)	187 (51.4%)
Body weight [kg] (SD)	80.78 ± 9.36
BMI [kg/m^2] (SD)	30.86 ± 4.16
FPG [mg/dL] (SD)	171 ± 26
PPPG [mg/dL] (SD)	262 ± 38
HbA1c [%]	8.4 ± 0.9
Median duration of diabetes [years] (SD)	6.63 ± 2.15
SBP [mmHg] (SD)	146 ± 10
DBP [mmHg] (SD)	92 ± 8
S. Cr. [mg/dL]	0.67 ± 0.2
Comorbidity, N (%)	
Dyslipidemia	147 (40.4%)
Hypertension	221 (60.7%)
Hypertension and dyslipidemia	82 (23.2%)
Concomitant anti-diabetes drug	
Metformin mono therapy	51 (14%)
Dual therapy	136 (37.4%)
Triple therapy	96 (26.4%)
Insulin therapy	81 (22.2%)

Data presented as mean ± SD or number (%)

BMI — body mass index; DBP — diastolic blood pressure; FPG — fasting plasma glucose; HbA1c — glycated hemoglobin; PPPG — postprandial plasma glucose; SBP — systolic blood pressure; S. Cr — serum creatinine

medication. Among the glycemic profile the mean HbA1c was 8.4 ± 0.9, FPG was 171 ± 26 mg/dL, and PPPG was 262 ± 38 mg/dL with a mean duration of diabetes of 6.63 ± 2.15 years. Mean systolic blood pressure was 146 ± 10 mmHg, and the mean diastolic blood pressure was 92 ± 8 mmHg. The most common comorbidity was hypertension (60.7%) followed by dyslipidemia (40.4%), and a large proportion of patients had both (23.2%) (Tab. 1).

Effectiveness

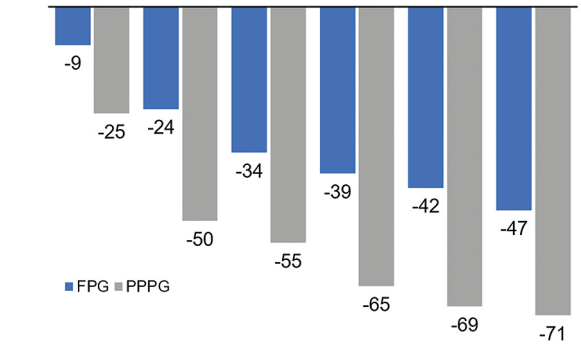
Based on ongoing glucose-lowering treatment the entire population was further divided into 4 groups (Tab. 2). Subjects who were on metformin monotherapy were classified under Group I [N = 51(14%)], subjects who were on duel therapy on metformin along with sulfonylureas/SGLT2i/DPP4i/alpha-glucosidase inhibitor were classified as Group II [N = 136 (37.4%)], subjects who were on triple drug therapy on metformin and sulfonylurea along with alpha-glucosidase inhibitor/DPP4i/SGLT2i were classified as Group III [N = 96 (26.4%)], and subjects who were on insulin therapy (premix insulin 45%, prandial insulin 23.8% and basal insulin 31.2%) were also grouped as group IV [N = 81 (22.2%)] (Tab. 2).

Patients were benefited with glycemic improvement from as early as the first week of the treatment period in conjunction with concurrent therapy and then maintained over 12 weeks (Fig. 1).

Table 2. Mean Reduction in Glycemic Parameters Post Administration of Lobeglitazone as an Add-On to Existing Glucose--Lowering Therapy at 12 Weeks (N = 364)

Concomitant glucose lowering agent	Glycemic parameters post administration of lobeglitazone as add-on								
	Baseline HbAic [%]	Δ HbA1c	p-value	Baseline FPG [mg/dL]	Δ FPG	p-value	Baseline PPPG [mg/dL]	Δ PPPG	p-value
Overall (N = 364)	8.4 ± 0.9	–1.1 ± 0.72	< 0.005	171 ± 26	–47.11 ± 21,45	< 0.005	262 ± 38	–71.47 ± 26.73	< 0.005
Group I (N = 51)	8.38± 0.8	–1.0 ± 0.68	< 0.005	166 ± 28	–45.12 ± 17.03	< 0.005	234 ± 26	–66.78 ± 25.42	< 0.005
Group II (N = 136)	8.2 ± 0.68	–1.1 ± 0.46	< 0.005	175 ± 25	–46.21 ± 16.59	< 0.005	292 ± 71	–90.54 ± 21.27	< 0.005
Group III (N = 96)	8.4 ± 0.92	–1.1 ± 0.64	< 0.005	165 ± 30	–51.09 ± 21.87	< 0.005	219 ± 68	–68.84 ± 19.56	< 0.005
Group IV (N = 81)	8.4 ± 0.87	–1.1 ± 0.59	< 0.005	178 ± 28	–49.95 ± 21.08	< 0.005	281 ± 48	–56.84 ± 28.52	< 0.005

Data presented as mean ± SD
HbA1c — glycated hemoglobin; FPG — fasting plasma glucose; PPPG — post prandial plasma glucose



	1st week	3rd week	5th Week	7th Week	9th Week	12th Week
FPG	-8,81	-23,64	-33,75	-38,61	-41,89	-47,11
PPPG	-24,68	-49,58	-54,63	-64,7	-69,42	-71,47

Figure 1. Mean Change (SD) in FPG and PPPG Levels (in mg/dL) from Baseline Post Administration of Lobeglitazone as an Add-On to Existing Glucose-Lowering Therapy over 12 Weeks (N = 364)
FPG — fasting plasma glucose; PPPG — post prandial plasma glucose

The primary end point was change in HbA1c reduction. The addition of lobeglitazone 0.5 mg to existing therapy over 12 weeks resulted in a statistically significant HbA1c reduction (–1.1 ± 0.72, p < 0.005) (Tab. 2). Among all 4 groups similar glycemic decrement was observed with no major intergroup variation (p = 0.074).

Regarding the secondary end point, a statistically significant mean reduction was also observed in both PPPG (–71.47 ± 26.73, p < 0.005) and FPG (–47.11 ± 21.45, p < 0.005), which was also similar across treatment groups (p = 0.95).

There was no significant correlation between background therapies and patient proportions achieving target HbA1c (< 7%) when, according to background therapy, we compared the probability of achieving the target. Details regarding the above are mentioned in Supplementary Table 1.

When based on baseline characteristics, the BMI was found to be significantly associated with achieving the recommended target HbA1c (57.3% in BMI 25–30 vs. 34% BMI > 30, p = 0.0233) as well as the target FPG (59% in BMI 25–30 verses 45.3% BMI > 30, p = 0.0112). Details regarding the above are mentioned in Supplementary Table 2.

There was no significant reduction in body weight over 12 weeks of additional treatment with lobeglitazone 0.5 mg once daily to existing glucose lowering therapy (–1.01 ± 0.94, p = 0.934). In insulin-treated patients, i.e., in Group IV, the weight loss was greater, indicating a beneficial effect of this combination with insulin therapy (–1.38 ± 0.93, p = 0.023). Overall, there was a significant drop in both the DBP (–3.18 ± 1.8, p = 0.121) and SBP (–7.65 ± 5.6, p = 0.063) in all participants. Serum Cr was unchanged throughout the treatment tenure across all the groups. Details regarding the above are mentioned in Supplementary Table 3.

Adding lobeglitazone showed a significant effect on plasma total cholesterol, triglyceride, and LDL cholesterol. Over 12 weeks of treatment the maximum reduction was observed in triglyceride (–34 ± 19, p = 0.07). Details regarding the above are mentioned in Supplementary Table 4.

Safety

Among the total population, 35 (9.61%) patients reported hypoglycemia. The maximum number of hypoglycemic events (20.9%) occurred in the insulin group followed by Group III (9.4%) (Tab. 3). In the insulin-treated group 3 cases of confirmed hypoglycemia (≤ 70 mg/dL) were reported. No patients having hypoglycemia required additional medical assistance, and overall hypoglycemia was moderate.

Table 3. Hypoglycemic Events Post Administration of Lobeglitazone as an Add-On to Existing Glucose-Lowering Therapy at 12 Weeks (N = 364)

Concomitant glucose lowering agent	Hypoglycemic events		
	Hypoglycemia (≤ 70 mg/dL)	Hypoglycemia (70–74 mg/d)	Total hypoglycemic events
Overall (N = 364)	4 (1%)	31 (8.5%)	35 (9.6%)
Group I (N = 51)	0	2 (3.9%)	2 (3.9%)
Group II (N = 136)	0	7 (5.1%)	7 (5.1%)
Group III (N = 96)	1 (1%)	8 (8.3%)	9 (9.4%)
Group IV (N = 81)	3 (3.7%)	14 (17.2%)	17 (20.9%)

Data presented as number (%)

Except for 3 patients with 15–20 WBCs in their urine at routine examination, there were no other symptoms or signs of urinary infection noted during the study period. 9.2% of patients were reported with genital mycotic infection, which was more common in females. Over 12 weeks drug-related AEs did not develop in any of the patients and no fatalities were reported. Supplementary Table 5 summarizes the undesirable effects that were observed.

Discussion

Due to the considerable heterogeneity in the diabetes population, in real-life settings, variations in outcomes may be observed as compared to randomized controlled trials (RCTs), which provide the highest level of evidence. Because there are limited data on lobeglitazone, which is a novel treatment option available since 2022 in India, the present study in more representative population settings was planned to investigate the effect on metabolic risk factors and glycemic improvements.

Lobeglitazone has been known to reduce blood sugar levels, lower HbA1c levels, and improve lipid and liver profiles by activating PPAR-γ and promoting the binding of insulin to fat cells [13, 14]. Lobeglitazone has similar effectiveness exhibited at a lower dose than pioglitazone, due to its higher affinity to PPAR-γ [13]. Besides exerting beneficial effects on diabetes, lobeglitazone also has favorable effects on other organs. In obese experimental models, lobeglitazone reduces hepatic steatosis by increasing insulin sensitivity and inhibiting hepatic lipogenesis. In *in vitro* or *in vivo* models with atherosclerosis, lobeglitazone significantly reduced neointimal formation after balloon injury in carotid arteries. Moreover, lobeglitazone had a good safety profile because it did not inhibit osteoblast differentiation *in vitro* and exerted no adverse effect on bone mineral density in experimental studies. Lobeglitazone was first approved in Korea in July 2013 for the management of type 2 diabetes [9], and recently in 2022 it was approved in India by the Drug Controller General of India.

To our best knowledge, this is the first real-world analysis of lobeglitazone 0.5 mg once daily to evaluate its safety and effectiveness in wide range of adult Indian patients with type 2 diabetes. In this retrospective observational study, over 12 weeks in suboptimally controlled patients in combination with existing antidiabetic therapy, lobeglitazone yielded a robust control of glycemic and metabolic parameters. Kim et al. [13] documented that monotherapy with lobeglitazone in patients with type 2 diabetes for 24 weeks resulted in a significant reduction in HbA1c versus placebo (–0.44% vs. 0.16%, mean difference –0.6%, $p < 0.0001$), and the effectiveness remained unchanged after 52 weeks of treatment [10]. In the present study lobeglitazone, when added to metformin, reduced HbA1c by –1.0 ± 0.68% ($p < 0.005$), and the results are in line with the observation by Jin et al. [15]. In a previous study, in patients with T2D as add-on treatments to metformin, when evaluating the role of different anti-diabetic agents, the addition of a TZD to metformin yielded the most durable glycemic response [16]. As compared to rosiglitazone and pioglitazone the binding affinity of lobeglitazone is 12-fold higher, which further results in enhanced binding affinity of lobeglitazone for PPAR-γ [17]. Although thiazolidinediones have been shown to act as peripheral insulin sensors [18], lobeglitazone has been shown to have positive effects on the survival and function of pancreatic β-cells in an animal research [19]. These factors may be responsible for the sustainable and long-term glycemic effectiveness with lobeglitazone. In our study, when lobeglitazone was added to existing double drug therapy, it resulted in an HbA1c reduction of –1.1 ± 0.64 ($p < 0.005$), and a similar result was also documented by Lim et al. [20] with metformin 1000 mg/day, sitagliptin 100 mg/day, and lobeglitazone 0.5 mg/day. According to a recent meta-analysis conducted by Joshi et al. [21], the safety profile of lobeglitazone was similar to that of a placebo, and it was well tolerated. According to this article, the HbA1C level changed by –0.23% (95% CI: –0.62 to 0.16) after receiving lobeglitazone.

A few studies, both randomized [13–15] and one real world [22], documented improvement in lipid profile with lobeglitazone. In line with those previous studies, the present study also documented a significant effect of lobeglitazone on plasma total cholesterol, triglyceride, and LDL cholesterol.

TZD is well-known for associated weight gain [23]. In some previous studies, treatment with lobeglitazone was responsible for increased body weight by 0.89 and 1.65 kg at 24 and 52 weeks, respectively [13, 14]. In the current study the weight loss (not statistically significant) may be due the effect of other previously used drugs. In insulin-treated patients, i.e., in Group IV, the weight loss was greater, indicating a beneficial effect of this combination with insulin therapy (–1.38 ± 0.93, $p = 0.023$). For further comment on weight neutrality, a large study is required. Edema and weight gain were comparable between half-maximal pioglitazone (15 mg/d) and lobeglitazone 0.5 mg/d according to a meta-analysis [24] of data from 26 trials including 828 people [RR 1.65 95% CI: 0.78–1.47). In all groups, the percentage increases in bone mineral density at the femur neck were similar [MD 0.07% (95% CI: 0.19–0.33); $p = 0.60$; $I^2 = 91\%$].

In a clinical setting, for 12 weeks of treatment, we assessed the hypoglycemic events as one of the major safety concerns and found that none of the patients having hypoglycemia required additional medical assistance, and overall hypoglycemia was moderate. In previous studies major events of hypoglycemia were also not reported [13, 14].

There were some limitations to this study. First of all, the selection bias might have affected the results due to its retrospective design. Second, there was potential risk with TZDs, such as increased risk of bone fractures, bladder cancer, CHF, etc. [25–30], which has not been documented in this study. Third, finding a surrogate for the counterfactual is more difficult in single group research because there is no direct, parallel, untreated comparator. The findings of the current study provide a meaningful contribution because there are limited convincing data available until the study is completed. To confirm the findings of the study, longer-term and mechanistic studies are needed.

Conclusions

To summarize, in a wide ranging population, the present study provides promising evidence of the suitability of use of lobeglitazone in addition to various coexisting background diabetes therapies showing beneficial clinical effects on glycemic and metabolic risk factors and, in the progressive course of diabetes, also supporting its distinctive place in the pharmacotherapeutic range of antidiabetic agents. In suboptimally controlled diabetes patients, in combination with one or more commonly prescribed antidiabetic drugs, lobeglitazone significantly improved the glycemic and non-glycemic measures and was well tolerated.

Article information

Data availability

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Author contributions

Arjun Baidya: conceptualization; formal analysis; methodology; writing — original draft; data collection. Rishad Ahmed: conceptualization; supervision; data collection, writing — review and editing. Mridul Bera: investigation; resources; formal analysis; data collection. Amit Gupta: methodology; writing — review and editing; Mridul Kumar Guha: writing — review and editing.

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Conflict of interest

The authors declare no conflict of interest.

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