International Journal of Science and Healthcare Research
[Indexed, Peer-Reviewed Journal]
Vol. 10; Issue: 4; Oct.-Dec. 2025
Website: ijshr.com
Original Research Article *ISSN: 2455-7587*

Comparative Study of Efficacy and Safety of Saroglitazar vs Pioglitazone in Patients of Type 2 Diabetes Mellitus

Santosh Kumar[1], Kumar Gaurav[1], Mukesh Kumar[1], Priyanka Kumari[1], Arun Kumar[1], Asha Singh [2]

[1]Tutor/Senior Resident, [2]Professor & Head;
Department of Pharmacology, Nalanda Medical College, Patna, Bihar, India

Corresponding Author: Arun Kumar

DOI: https://doi.org/10.52403/ijshr.20250412

ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is highly prevalent in India, with patients often presenting with both insulin resistance and atherogenic dyslipidemia. Pioglitazone, a selective PPAR-γ agonist, improves insulin sensitivity but is limited by adverse effects such as weight gain and edema. Saroglitazar, a novel dual PPAR-α/γ agonist with predominant PPAR-α activity, has shown promise in improving both glycemic and lipid parameters with a more favorable safety profile. Comparative data between these two agents in Indian patients remain limited.

Methods: A prospective, randomized, open-label, comparative clinical trial was conducted over 12 months. A total of 120 patients with T2DM (HbA1c 7.0–9.0%) were randomized into two groups: Group S (Saroglitazar 4 mg once daily, n=60) and Group P (Pioglitazone 15 mg once daily, n=60). The primary endpoint was change in HbA1c at 12 months. Secondary endpoints included changes in fasting blood sugar (FBS), post-prandial blood sugar (PPBS), body weight, and incidence of adverse events.

Results: Both groups demonstrated significant reductions in HbA1c, FBS, and PPBS over 12 months ($p<0.0001$). However, Saroglitazar achieved significantly greater reductions in HbA1c at 6 and 12 months ($p=0.0350$ and $p=0.0461$), lower FBS and lower PPBS at 6 and 12 months ($p=0.0002$, <0.0001). Adverse events were more frequent in the Pioglitazone group, with significantly higher incidence of edema (10 vs. 2, $p=0.0295$) and a trend toward greater weight gain (12 vs. 4, $p=0.0575$).

Conclusion: Saroglitazar demonstrated superior long-term glycemic control and a more favorable safety profile compared to Pioglitazone in Indian patients with T2DM.

Keywords: Type 2 Diabetes Mellitus, Saroglitazar, Pioglitazone, PPAR agonist, Glycemic control, Dyslipidemia, Randomized clinical trial

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance, impaired insulin secretion, and progressive β-cell dysfunction. [(1)] It is associated with multiple comorbidities, including dyslipidemia, hypertension, and cardiovascular disease, which together contribute to significant morbidity and mortality worldwide. [(1)] India is often referred to as the "diabetes capital of the world," with an estimated 77 million adults living with diabetes as of 2019, a number

projected to rise to over 100 million by 2030. [2]

Over the past decades, pharmacological management of T2DM has expanded beyond traditional agents such as sulfonylureas and metformin to include thiazolidinediones, DPP-4 inhibitors, GLP-1 receptor agonists, and SGLT2 inhibitors. Among these, pioglitazone, a thiazolidinedione, has been widely used for its insulin-sensitizing effects mediated through peroxisome proliferator-activated receptor gamma (PPAR-γ) activation. [3]

More recently, saroglitazar, a novel dual PPAR-α/γ agonist, has been introduced in India with the promise of addressing both glycemic control and diabetic dyslipidemia. [4] Given the dual burden of hyperglycemia and atherogenic dyslipidemia in Indian patients, comparative evaluation of these two agents is of particular clinical relevance. [5]

A distinctive feature of T2DM in the Indian population is its early onset, often a decade earlier than in Western populations, and its strong association with central obesity, insulin resistance, and dyslipidemia. [2, 5] The so-called "Asian Indian phenotype" is characterized by higher visceral adiposity, lower lean body mass, and a predisposition to atherogenic dyslipidemia, even at lower body mass indices. [2, 5]

Pioglitazone, a selective PPAR-γ agonist, improves insulin sensitivity in adipose tissue, skeletal muscle, and the liver. It enhances glucose uptake, reduces hepatic gluconeogenesis, and exerts favorable effects on lipid metabolism by modestly increasing HDL-C and lowering triglycerides. However, its use is limited by adverse effects such as weight gain, fluid retention, risk of heart failure, and concerns regarding long-term safety. [6]

Saroglitazar, in contrast, is a dual PPAR-α/γ agonist with predominant PPAR-α activity and moderate PPAR-γ activity. This pharmacological profile allows it to exert insulin-sensitizing effects while also significantly improving lipid parameters, particularly hypertriglyceridemia. [7] Clinical studies have demonstrated that saroglitazar reduces triglycerides, non-HDL cholesterol, and improves glycemic indices such as HbA1c and fasting plasma glucose. Importantly, its safety profile appears favorable, with lower incidence of weight gain and edema compared to pioglitazone. [8]

The comparative pharmacology of these two agents highlights a potential therapeutic advantage of saroglitazar in Indian patients, who often present with the dual burden of insulin resistance and dyslipidemia. However, head-to-head comparative data remain limited, necessitating systematic evaluation.

There is a paucity of robust randomized controlled trials directly comparing the efficacy and safety of saroglitazar versus pioglitazone in Indian patients with T2DM. Given the unique epidemiological and phenotypic characteristics of the Indian diabetic population, extrapolation of global data may not be appropriate. A comparative study is therefore essential to determine whether saroglitazar offers superior efficacy and safety in this context, and whether it can be considered a viable alternative to pioglitazone in routine clinical practice.

The rationale for this study lies in addressing these evidence gaps by systematically evaluating the comparative efficacy and safety of saroglitazar and pioglitazone in Indian patients with T2DM. The present study seeks to address the research question: *In Indian patients with Type 2 Diabetes Mellitus, does saroglitazar demonstrate superior efficacy and safety compared to pioglitazone over a 12-month treatment period?* Accordingly, the objectives of this study are threefold: (1) to compare the efficacy of saroglitazar and pioglitazone in reducing HbA1c as the primary endpoint; (2) to evaluate their effects on secondary glycemic parameters including FBS and PPBS, as well as on body weight; and (3) to assess and contrast the safety profiles of both agents by documenting and analyzing the incidence of adverse events during the study period.

MATERIALS & METHODS

Study Overview: This study was designed as a prospective, randomized, open-label, comparative clinical trial conducted over a period of 12 months at a tertiary care teaching hospital in India. The study adhered to the principles of the Declaration of Helsinki and Good Clinical Practice guidelines, and ethical approval was obtained from the Institutional Ethics Committee prior to initiation. Written informed consent was obtained from all participants.

Study Population: Patients eligible for inclusion in the study were adults aged 30–65 years with a confirmed diagnosis of Type 2 Diabetes Mellitus (T2DM) according to American Diabetes Association (ADA) criteria, [9] who had HbA1c levels between 7.0% and 9.0% at baseline despite being on stable doses of standard oral antidiabetic drugs for at least three months. Patients were excluded if they had Type 1 diabetes mellitus or secondary forms of diabetes, a history of cardiovascular events such as myocardial infarction, stroke, or heart failure within the past six months, significant hepatic impairment (ALT/AST >3 times the upper limit of normal) or renal impairment (eGFR <45 mL/min/1.73 m²), or a history of bladder cancer, macular edema, or active malignancy. Pregnant or lactating women, those planning pregnancy during the study period, and individuals with known hypersensitivity to either saroglitazar or pioglitazone were also excluded.

Sample Size

The sample size was calculated based on the assumption of detecting a minimum clinically significant difference of 0.5% in HbA1c reduction between the two groups, with a power of 80% and a two-sided alpha of 0.05. Accounting for a 10% dropout rate, a total of 120 patients were recruited, with 60 patients allocated to each treatment arm.

Outcome Parameters

- **Primary Endpoint:** Reduction in mean HbA1c from baseline to 12 months.
- **Secondary Endpoints:** Changes in fasting blood sugar (FBS), post-prandial blood sugar (PPBS), body weight, and incidence of adverse events during the study period.

Data Collection

Baseline demographic data, medical history, and clinical characteristics were recorded at enrollment. Laboratory investigations including HbA1c, FBS, PPBS, lipid profile, liver function tests, and renal function tests were performed at baseline and at 3, 6, and 12 months. Body weight and blood pressure were measured at each follow-up visit. Adverse events were documented at every visit through patient interviews and clinical examination. Compliance with medication was assessed by pill count and patient self-reporting. [10]

METHODOLOGY

Eligible patients were randomized using a computer-generated randomization sequence into two groups:

- Group S: Saroglitazar 4 mg once daily.
- Group P: Pioglitazone 15 mg once daily.

Both groups continued their background therapy with metformin and/or sulfonylureas as per treating physician's discretion, provided doses remained stable throughout the study. Lifestyle advice regarding diet and physical activity was reinforced at each visit. Patients were followed up at 3-month intervals for clinical assessment, laboratory investigations, and monitoring of adverse events. Dose adjustments of background therapy were permitted only if clinically indicated, and such changes were documented.

Statistical Analysis

Data were analyzed using Graph Pad Version 8.4.3. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Between-group comparisons of continuous variables were performed using the independent samples t-test. Within-group comparisons from

baseline to follow-up were analyzed using repeated measure ANOVA. Categorical variables were compared using the chi-square test or Fisher's exact test. A p-value of <0.05 was considered statistically significant.

RESULTS

60 patients were enrolled in pioglitazone group (Group P) and 60 patients were enrolled in saroglitazar group (Group S). All baseline variables were well-balanced between the two groups, as indicated by non-significant p-values (ranging from 0.6994 to 0.8772), suggesting that both groups were comparable at the start of the study, thereby minimizing potential confounding factors in the analysis of treatment outcomes [Table 1].

Table 1: Comparison of Baseline Demographic and Clinical Characteristics between Pioglitazone Group (Group P) and Saroglitazar Group (Group S)

Parameters	Group P (n = 60)	Group S (n = 60)	P-Value
Age in years (Mean ± SD)	59.47 ± 7.21	58.93 ± 8.05	0.6994*
Number of Females (%)	35 (58.33)	32 (53.33)	0.7133**
Duration of T2DM in Years (Mean ± SD)	6.98 ± 3.36	7.17 ± 3.92	0.7761*
Body Weight in kg (Mean ± SD)	62.85 ± 10.74	63.20 ± 11.39	0.8628*
BMI in kg/m^2 (Mean ± SD)	25.22 ± 3.41	25.47 ± 4.02	0.7140*
LDL in mg/dl (Mean ± SD)	152.41 ± 21.69	153.06 ± 20.75	0.8772*
*Unpaired t-test **Fisher's exact test			

While both groups showed significant reductions in HbA1c over time ($p < 0.0001$ for both, per ANOVA), significant between-group differences emerged at 6 and 12 months, with Group S demonstrating lower mean HbA1c levels ($p = 0.0350$ and $p = 0.0461$, respectively). This indicates that Saroglitazar may offer superior long-term glycemic control compared to Pioglitazone [Table 2] [Figure 1].

Table 2: Comparison of HbA1c Levels between Pioglitazone Group (Group P) and Saroglitazar Group (Group S)

Time	Mean HbA1c (%) in Group P ± SD	Mean HbA1c (%) in Group S ± SD	P-Value (Unpaired t-test)
Baseline	8.05 ± 1.11	8.08 ± 1.09	0.8815
3 Months	7.75 ± 1.30	7.44 ± 1.32	0.1975
6 Months	7.57 ± 1.16	7.15 ± 0.99	0.0350
12 Months	7.33 ± 0.99	6.99 ± 0.93	0.0461
P-Value (ANOVA)	<0.0001	<0.0001	

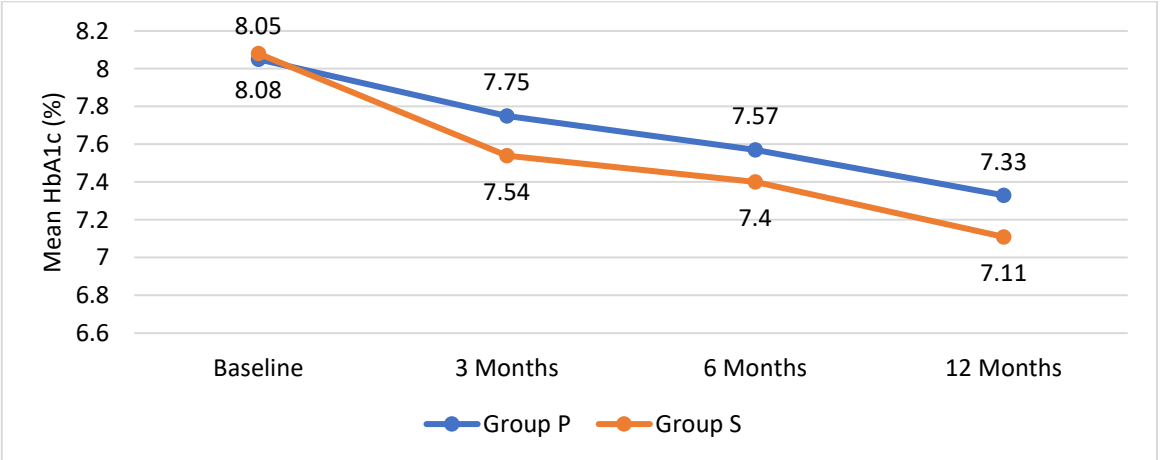

Figure 1: Comparison of Mean HbA1c between Pioglitazone Group (Group P) and Saroglitazar Group (Group S)

Both groups experienced significant reductions in FBS over time (ANOVA $p < 0.0001$). However, Group S consistently achieved lower FBS levels than Group P from 3 months onward, with statistically significant differences at all follow-up points ($p = 0.0368$ at 3 months, $p = 0.0013$ at 6 months, and $p = 0.0063$ at 12 months), suggesting more effective fasting glucose management with Saroglitazar [Table 3].

Table 3: Comparison of Mean Fasting Blood Sugar at Different Follow-up between Pioglitazone Group (Group P) and Saroglitazar Group (Group S)

Time	Mean FBS (mg/dl) in Group P ± SD	Mean FBS (mg/dl) in Group S ± SD	P-Value (Unpaired t-test)
Baseline	176.59 ± 25.27	177.13 ± 26.36	0.8989
3 Months	161.80 ± 25.21	153.39 ± 18.33	0.0368
6 Months	151.74 ± 20.56	140.43 ± 16.94	0.0013
12 Months	140.36 ± 20.65	131.29 ± 14.71	0.0063
P-Value (ANOVA)	<0.0001	<0.0001	

Table 4: Comparison of Mean Post-prandial Blood Sugar at Different Follow-up between Pioglitazone Group (Group P) and Saroglitazar Group (Group S)

Time	Mean PPBS (mg/dl) in Group P ± SD	Mean PPBS (mg/dl) in Group S ± SD	P-Value (Unpaired t-test)
Baseline	236.71 ± 33.28	237.07 ± 31.24	0.9595
3 Months	214.80 ± 30.11	204.64 ± 26.82	0.0524
6 Months	200.86 ± 29.66	181.44 ± 24.50	0.0002
12 Months	183.48 ± 27.56	162.58 ± 18.81	<0.0001
P-Value (ANOVA)	<0.0001	<0.0001	

Both treatments significantly reduced PPBS over 12 months (ANOVA $p < 0.0001$). Saroglitazar (Group S) showed markedly lower PPBS levels compared to Pioglitazone (Group P) at 6 and 12 months ($p = 0.0002$ and $p < 0.0001$, respectively), and a strong trend at 3 months ($p = 0.0524$), indicating better post-meal glucose control with Saroglitazar over the long term [Table 4].

A significantly higher incidence of edema was observed in Group P compared to Group S ($p = 0.0295$). Weight gain was also more frequent in Group P, though not statistically significant ($p = 0.0575$). Other adverse events such as headache, myalgia, upper respiratory tract infection, and sinusitis were numerically higher in Group P but did not reach statistical significance, suggesting a potentially better safety profile for Saroglitazar, particularly regarding oedema [Table 5].

Table 5: Comparison of Incidence of Adverse Events between Lobeglitazone Group and Pioglitazone Group

Parameters	Group P (n = 60)	Group S (n = 60)	P-Value (Fisher's exact test)
Oedema	10	2	0.0295
Weight Gain	12	4	0.0575
Headache	5	1	0.2068
Myalgia	3	0	0.2437
Upper respiratory tract infection	4	1	0.3644
Sinusitis	2	0	0.4958

DISCUSSION

The study compares Pioglitazone, a classic insulin-sensitizing thiazolidinedione (TZD), and Saroglitazar, a newer dual peroxisome proliferator-activated receptor (PPAR) agonist. Their mechanisms of action are key to interpreting the results. Pioglitazone is primarily a PPAR-γ agonist. Its activation improves insulin sensitivity in adipose, muscle, and liver tissue by promoting adipocyte differentiation and reducing circulating free fatty acids. However, its

strong PPAR-γ agonism is also linked to classic side effects like weight gain and fluid retention (edema). [3] Saroglitazar is a dual PPAR-α/γ agonist with a predominant PPAR-α activity. PPAR-α activation (like fibrates) primarily regulates lipid metabolism, lowering triglycerides and increasing HDL-C. The concomitant, but modulated, PPAR-γ activation provides glycemic control. This unique profile aims to deliver the insulin-sensitizing benefits of a TZD while leveraging the potent lipid-lowering effects of a fibrate, potentially mitigating the adverse effects associated with strong PPAR-γ activation. [4]

The results of our study demonstrate a clear and growing therapeutic advantage for Saroglitazar over Pioglitazone over a 12-month period. While both drugs significantly improved glycemic parameters from baseline (significant ANOVA p-values), Saroglitazar (Group S) consistently outperformed Pioglitazone (Group P). The significantly lower HbA1c at 6 and 12 months, along with significantly lower Fasting and Post-prandial Blood Sugar levels from 3 months onward, indicates that Saroglitazar provides not just equivalent, but superior glycemic control in the long term. This suggests its dual mechanism may offer a more comprehensive approach to managing hyperglycemia.

The adverse event data is clinically crucial. The significantly higher incidence of edema in the Pioglitazone group (10 vs. 2, p=0.0295) and a strong trend towards more weight gain (12 vs. 4, p=0.0575) align perfectly with the known side-effect profile of strong PPAR-γ agonists. The markedly lower rates of these events with Saroglitazar suggest its modified PPAR-γ activity offers a much-improved tolerability, which is a major factor in long-term medication adherence and patient quality of life.

The findings of our study are strongly reinforced by and consistent with the existing body of evidence on Saroglitazar. Our finding that Saroglitazar leads to significantly greater reductions in HbA1c and FPG/PPBS aligns with multiple studies. Krishnappa et al. (2020) and Sreekumar et al. (2021) found comparable and significant HbA1c reductions between Saroglitazar and Pioglitazone. [11, 12] Our study extends this by showing a statistically significant superiority at later time points. This is corroborated by the meta-analysis of Simental Mendía et al. (2025), which confirms significant weighted mean differences for FPG, PPBS, and HbA1c in favor of Saroglitazar. [13] Furthermore, Jain et al. (2019) provides a mechanistic insight, showing that these glycemic benefits are likely driven by improved insulin sensitivity and β-cell function. [14]

While our analysis does not include lipid outcomes, the established mechanism of Saroglitazar and the consistent results from the previous studies form a critical part of its clinical value. The PRESS V (Pai et al., 2014), PRESS VI (Jani et al., 2014), and PRESS XII (Krishnappa et al., 2020) trials overwhelmingly demonstrate Saroglitazar's superior efficacy in reducing triglycerides, LDL-C, VLDL-C, and non-HDL-C compared to Pioglitazone. [11, 15, 16] This dual benefit is its defining characteristic, addressing both dysglycemia and atherogenic dyslipidemia—a common and dangerous combination in T2DM known as "residual cardiovascular risk." [17]

Our adverse event findings are a cornerstone of Saroglitazar's value proposition and are strongly supported by previous literature. The lower rates of edema and weight gain you observed are consistent with the drug's pharmacologic profile. Most of the cited studies (Pai et al., 2014; Krishnappa et al., 2020; Sreekumar et al., 2021; Jani et al., 2014) report that Saroglitazar is "well-tolerated" with no major safety signals, [11, 12, 15, 16]) and real-world data from Chatterjee et al. (2015) confirmed significant metabolic benefits without significant weight change. [18]

The positive results in our T2DM population are echoed in other patient groups. Dhone et al. (2022) showed significant HbA1c and triglyceride reductions in prediabetic patients, suggesting a potential role for

saroglitazar in early intervention for cardio-metabolic risk. [19, 20]

A key limitation of this study is its single-center design and relatively small sample size (n=60 per group), which may limit the generalizability of the findings to broader, more diverse populations. The focus on glycemic parameters without reporting lipid outcomes also prevents a comprehensive evaluation of saroglitazar's full therapeutic potential, which is known to include significant lipid-lowering effects.

CONCLUSION

Our 12-month study robustly confirms that Saroglitazar offers a superior clinical profile compared to Pioglitazone. It provides significantly better long-term glycemic control (HbA1c, FBS, PPBS) and a significantly more favorable safety and tolerability profile, particularly regarding edema and weight gain. When integrated with the previous research, it becomes evident that saroglitazar's unique dual PPAR-α/γ agonism successfully decouples potent glycemic and lipid-lowering efficacy from the typical TZD-related adverse effects. This makes it a highly compelling therapeutic option, especially for T2DM patients with co-existing dyslipidemia, where it can address two critical cardiovascular risk factors simultaneously.

Declaration by Authors

Ethical Approval: Approved

Acknowledgement: None

Source of Funding: None

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

REFERENCES

1. Arya P, Husain N, Kumar C, Shekhar R, Prakash V, Hameed S, Mohan L, Dikshit H. C-peptide Level in Patients with Uncontrolled Type 2 Diabetes Mellitus on Oral Anti-diabetic Drugs. *Cureus.* 2024 Mar 24;16(3):e56810. doi: 10.7759/cureus.56810.
2. Jha RP, Shri N, Patel P, Dhamnetiya D, Bhattacharyya K, Singh M. Trends in the diabetes incidence and mortality in India from 1990 to 2019: a joinpoint and age-period-cohort analysis. *J Diabetes Metab Disord.* 2021 Jul 5;20(2):1725-1740. doi: 10.1007/s40200-021-00834-y.
3. Basak S, Murmu A, Matore BW, Roy PP, Singh J. Thiazolidinedione an auspicious scaffold as PPAR-γ agonist: its possible mechanism to manoeuvre against insulin resistant diabetes mellitus. *Eur J Med Chem Rep.* 2024 Aug; 11:100160. doi: 10.1016/j.ejmcr.2024.100160
4. Das S, Gupta S, Lathia T, et al. Evaluation of Effectiveness and Tolerability of Saroglitazar in Metabolic Disease Patients of India: A Retrospective, Observational, Electronic Medical Record-Based Real-World Evidence Study. *Cureus.* 2025 Jul 30;17(7): e89028. doi: 10.7759/cureus.89028
5. Lone SS, Majid S, Bhat MH, Wani GA. Exploring the burden of mixed dyslipidemia in patients with type 2 diabetes mellitus: A cross-sectional study in Kashmir, India. *Am J Hum Biol.* 2024 Apr;36(4):e24012. doi: 10.1002/ajhb.24012.
6. Singh G, Can AS, Correa R. Pioglitazone. [Updated 2023 Jul 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK544287/
7. Jain MR, Giri SR, Trivedi C, Bhoi B, Rath A, Vanage G, Vyas P, Ranvir R, Patel PR. Saroglitazar, a novel PPARα/γ agonist with predominant PPARα activity, shows lipid-lowering and insulin-sensitizing effects in preclinical models. *Pharmacol Res Perspect.* 2015 Jun;3(3):e00136. doi: 10.1002/prp2.136.
8. Sharma A, Amarnath S, Kushwah DS, Ramaswamy S. Saroglitazar, a novel cardiometabolic agent for diabetic dyslipidemia – A review. *J Young Pharm.* 2015;7(1):2-6. doi:10.5530/jyp.2015.1.2.
9. American Diabetes Association Professional Practice Committee. 2. Diagnosis and classification of diabetes: Standards of care in diabetes—2024. *Diabetes Care.* 2024 Jan 1;47(Suppl 1):S20-42. doi:10.2337/dc24-S002.
10. Stirratt MJ, Dunbar-Jacob J, Crane HM, et al. Self-report measures of medication adherence behavior: recommendations on optimal use. *Transl Behav Med.* 2015 Dec;5(4):470-82. doi: 10.1007/s13142-015-0315-2.

11. Krishnappa M, Patil K, Parmar K, Trivedi P, Mody N, Shah C, Faldu K, Maroo S; PRESS XII study group; Parmar D. Effect of saroglitazar 2 mg and 4 mg on glycemic control, lipid profile and cardiovascular disease risk in patients with type 2 diabetes mellitus: a 56-week, randomized, double blind, phase 3 study (PRESS XII study). *Cardiovasc Diabetol.* 2020 Jun 19;19(1):93. doi: 10.1186/s12933-020-01073-w.
12. Sreekumar V, Meher D, Goutam S, Mishra MR, Tripathy R, Jena J. Effect of saroglitazar and pioglitazone on lipid parameters and glycemic profile in type II diabetes mellitus patients with dyslipidemia: An observational open-label study. *Natl J Physiol Pharm Pharmacol.* 2021; 11(5): 514-518. doi:10.5455/njppp.2021.11.0100620211201 2021
13. Simental-Mendía LE, Simental-Mendía M, Barragán-Zuñiga LJ, Arce-Quiñones M. Effect of saroglitazar on glycaemic parameters: A systematic review and meta-analysis of randomized controlled trials. *Diabetes Obes Metab.* 2025 Sep;27(9):4627-4642. doi: 10.1111/dom.16506.
14. Jain MR, Giri SR, Trivedi C, et al. Saroglitazar, a novel PPARα/γ agonist with predominant PPARα activity, shows lipid-lowering and insulin-sensitizing effects in preclinical models. *Pharmacol Res Perspect.* 2015 Jun;3(3):e00136. doi: 10.1002/prp2.136.
15. Pai V, Paneerselvam A, Mukhopadhyay S, et al. A Multicenter, Prospective, Randomized, Double-blind Study to Evaluate the Safety and Efficacy of Saroglitazar 2 and 4 mg Compared to Pioglitazone 45 mg in Diabetic Dyslipidemia (PRESS V). *J Diabetes Sci Technol.* 2014 Jan;8(1):132-141. doi: 10.1177/1932296813518680.
16. Jani RH, Pai V, Jha P, Jariwala G, Mukhopadhyay S, Bhansali A, Joshi S. A multicenter, prospective, randomized, double-blind study to evaluate the safety and efficacy of Saroglitazar 2 and 4 mg compared with placebo in type 2 diabetes mellitus patients having hypertriglyceridemia not controlled with atorvastatin therapy (PRESS VI). *Diabetes Technol Ther.* 2014 Feb;16(2):63-71. doi: 10.1089/dia.2013.0253.
17. Ansar S, Koska J, Reaven PD. Postprandial hyperlipidemia, endothelial dysfunction and cardiovascular risk: focus on incretins. *Cardiovasc Diabetol.* 2011 Jul 7; 10:61. doi: 10.1186/1475-2840-10-61.
18. Chatterjee S, Majumder A, Ray S. Observational study of effects of saroglitazar on glycaemic and lipid parameters on Indian patients with type 2 diabetes. *Sci Rep.* 2015; 5:7706. doi:10.1038/srep07706
19. Dhone PG, Behera D, Gupta V, Mubeen MF. To evaluate the safety and efficacy of saroglitazar among pre-diabetes and dyslipidemia. *Int J Health Sci.* 2022;6(S2):8835. doi:10.53730/ijhs.v6nS2.8835
20. Das S, Gupta S, Lathia T, et al. Evaluation of effectiveness and tolerability of saroglitazar in metabolic disease patients of India: a retrospective, observational, electronic medical record-based real-world evidence study. *Cureus.* 2025 Jul 30;17(7):e89028. doi:10.7759/cureus.89028

How to cite this article: Santosh Kumar, Kumar Gaurav, Mukesh Kumar, Priyanka Kumari, Arun Kumar, Asha Singh. Comparative study of efficacy and safety of Saroglitazar vs Pioglitazone in patients of type 2 diabetes mellitus. *Int. J. Sci. Healthc. Res.* 2025; 10(4): 76-83. DOI: *10.52403/ijshr.20250412*

******