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Long Term Safety and Efficacy of Saroglitazar in Indian Patients with Diabetic Dyslipidemia and Very High Triglyceride Levels: Real World Evidence

ABSTRACT

Objective: Diabetes and diabetic dyslipidemia with high triglycerides (TGs) are commonly associated. Saroglitazar is the first dual PPAR α/γ agonist approved in India for the management of diabetic dyslipidemia. The objective of this observational study was to assess the long-term safety and efficacy of saroglitazar in patients with diabetic dyslipidemia with very high triglycerides (> 500 mg/dL).

Materials and methods: This was a single-center, retrospective observational study which was conducted among 150 patients with type 2 diabetes who had diabetic dyslipidemia with hypertriglyceridemia (> 500 mg/dL at baseline). All patients were on saroglitazar 4 mg once daily and all had complete follow-up data. At baseline, all patients were on stable doses of antidiabetic and statin therapy.

Results: Significant reduction of TG and LDL-cholesterol was observed from baseline to 12th week: from 669.93 ± 81.22 to 268.72 ± 82.32 mg/dL and from 167.68 ± 10.881 to 118.88 ± 12.16 mg/dL ($p < 0.0001$ and < 0.001), respectively. The mean HbA1c was reduced from 8.02 ± 0.3 to 7.71 ± 0.5% (< 0.001). This reduction in lipid and glycemic parameters continued till 52th week. At 52 weeks mean TG, LDL-cholesterol and HbA1c were reduced to 221.51 ± 61.81 mg/dL, 118.88 ± 12.16 mg/dL and 7.12 ± 0.2 % ($p < 0.001$ for all). No major adverse event was reported during the study period. Creatine phosphokinase (CPK), liver enzymes and creatinine did not alter significantly.

Conclusions: Long term use of saroglitazar leads to significant improvement in both glycemic and lipid parameters in patients with diabetic dyslipidemia with very high triglycerides (>500 mg/dl) and initially not controlled by statins alone. Therefore, to fulfil the unmet need, long-term use of saroglitazar could be an effective, well tolerated and safe option. (Clin Diabetol 2022, 11; 5: 316–320)

Keywords: saroglitazar, high triglyceride, type 2 diabetes, dyslipidemia

Introduction

Dyslipidemia is one of the major concerns for people with diabetes and 75% of patients with diabetes have associated dyslipidemia [1]. The burden of cardiovascular diseases (CVDs) is greatly influenced by diabetic dyslipidemia (DD) and considered as one of the major contributing risk factors [2]. Another name of diabetic dyslipidemia is atherogenic dyslipidemia, which is a triad of high triglycerides (TG) and small dense low-density lipoprotein cholesterol (sd-LDL-C), and low high-density lipoprotein cholesterol (HDL-C)

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[3]. Worldwide, the most common cause of mortality is CVDs and the magnitude of this burden is growing rapidly [4]. As per INTERHEART study, among the 9 easily measured risk factors that account for over 90% of the risk of acute myocardial infarction (AMI), which include dyslipidemia, diabetes, hypertension, obesity, diet, smoking, alcohol consumption, physical activity and psychosocial factors, the strongest risk predictor of AMI is dyslipidemia [5]. Hypertriglyceridemia is one of the major risk factors of pancreatitis and it accounts for 1 to 4% of all cases of acute pancreatitis. When the triglyceride level is more than 500 mg/dL it can cause pancreatitis, but it becomes clinically significant when triglyceride levels are >1000 mg/dL [6–8]. When metabolic syndrome is treated and various components of dyslipidemia, especially triglycerides, are lowered, the rate of coronary events is reduced. High doses of omega-3 fatty acids and fibrates are drugs of choice for the initiation of pharmacological treatment after lifestyle modification. When hypertriglyceridemia is accompanied by elevated low-density lipoprotein levels, 3-hydroxy-3 methyl-glutaryl coenzyme A (HMG-CoA) reductase inhibitors (statins) are preferred. In patients with hypertriglyceridemia who have low HDL levels, extended release niacin can be considered.

The function of peroxisome proliferator-activated receptor-alpha (PPAR-alpha), which is mainly expressed in skeletal muscle, heart and liver, is to regulate fatty acid oxidation and ketone body synthesis. On the other hand, PPAR-gamma promotes triglyceride storage and differentiation of preadipocytes into adipocytes [9] and is present in lower intestine and adipose tissue. The only approved dual PPAR α/γ agonist for the treatment of diabetic dyslipidemia in India is saroglitazar. It has a positive effect on insulin sensitivity as well as a reducing effect on elevated TG. Addition of saroglitazar to statin led to a significant reduction in non-HDL cholesterol (−32.5%) and TG (−46.7%) in multiple randomized clinical trials [10, 11]. In patients with diabetic dyslipidemia, treatment with saroglitazar effectively reduces small-dense low-density lipoprotein (sd-LDL), non-high--density lipoprotein cholesterol (non-HDL-C) particles and triglycerides along with blood glucose level [12]. The present observational study was done to evaluate the long-term safety and efficacy of saroglitazar in Indian DD patients in real-world scenario.

Materials and methods

The present study is a single-center, retrospective observational study which was conducted among type 2 diabetes patients who were also diagnosed with diabetic dyslipidemia along with hypertriglyceridemia. The inclusion criteria of the study were patients with type 2 diabetes aged >18 years and having high triglycerides (> 500 mg/dL) and on antidiabetic drugs and statins. The exclusion criteria for the present study were type 1 diabetes mellitus, moderate or severe liver and kidney disease, patients receiving fenofibrate and pioglitazone within previous 3 months, documented heart failure (NYHA class III or IV) and having any type of malignancy. As the lipid parameters of the participants were not controlled with statin alone, saroglitazar 4 mg was added as add-on therapy as per the approved indication by DCGI (Drugs Controller General of India). Pre-designed questionnaires were used to collect data of patients who visited the clinic from January 2014 to November 2018 and had been prescribed saroglitazar.

Initially, 164 patients with documented hypolipidemic and antidiabetic medication history and who were prescribed saroglitazar 4 mg tablet once daily before breakfast at baseline were selected for this study. Finally, data of 150 patients were selected after excluding 4 patients who did not have proper antidiabetic medication history and 10 patients who didn't continue saroglitazar beyond 3 months. For each patient the analysis was done for three visits including at baseline, 3 months and 12 months. Glycemic parameters for the analysis were fasting plasma glucose, post-prandial plasma glucose and glycated hemoglobin (HbA1c); and lipid parameters were low-density lipoprotein (LDL), high-density lipoprotein (HDL), total cholesterol (TC), triglyceride (TG) and non-high-density lipoprotein cholesterol (non-HDL-C). Pathological test reports of NABL (National Accreditation Board for Testing and Calibration Laboratories), accredited laboratories preferred by patients, were considered for the analysis. The LDL-C values and non-HDL-C were measured by direct method.

To compare the demographic and baseline disease characteristics descriptive statistics was used. For continuous variables data were presented as mean ± SD, median, percentiles or range and for categorical variables percentage was calculated. For homogeneity testing data from all the patients were compared at baseline using ANOVA test for continuous variables and Chi-square test or Fisher's exact test for categorical variable. For statistical analysis, SAS 9.3 were used and 0.05 was set as significance level for all statistical tests.

Results

One hundred fifty patients who received saroglitazar 4 mg once daily and continued for 12 months were included in the analysis. The mean age of the patients was 57.6 (± 8.1) years and 61.3% of the patients were male. Average duration of diabetes was 7.8 (± 2.4) years. The patients had a mean weight of 71.3 (± 10.35) kg and a mean body mass index of 26.8 (± 3.89) kg/m^2.

A history of smoking and alcoholism were present in 14.6% and 2.6% patients, respectively (Tab. 1). In this study, all the of patients were on statin therapy at baseline, with atorvastatin being the most commonly used statin (59.4%) (Tab. 1). All patients were advised to continue on-going statin therapy and saroglitazar 4 mg once daily was prescribed as 2nd line lipid-lowering agent. Of the 150 patients, 5.3% was on single antidiabetic therapy, 31.3% was on dual antidiabetic therapy and 63.3% was on more than two antidiabetic drugs (Tab. 1). In the study population, the most commonly reported antidiabetic drug at baseline was metformin in 96.6% of the patients, followed by sulfonylureas in 60.6%, dipeptidyl peptidase-4 (DPP4) inhibitors in 54.3%, alpha glucosidase inhibitors in 15.3%, insulin in 12.0%, sodium-glucose cotransporter-2 (SGLT2) inhibitor in 8.6%, hydroxychloroquine in 7.3%, glucagon-like peptide 1 (GLP1) agonist in 1.3% (Tab. 1).

Saroglitazar in addition to oral antidiabetic medication and statin therapy showed significant improvement in all lipid and glycemic parameters at 12-week follow up and that improvement continued till 52 weeks. The mean TG was 669.93 ± 81.22 mg/dL vs. 221.51 ± 61.81 mg/dL at baseline and 52-week follow-up, a significant reduction of 66.9% from baseline. A statistically significant change in all other lipid parameters was also noted (Fig. 1).. A mean reduction of 38.47% in LDL-C levels, 52.62% in non-HDL-C levels, 44.84% in total cholesterol levels and mean increase of 4.72% in HDL-C levels was noted. Analysis of glycemic parameters revealed a statistically significant 11.22% reduction (0.9% point absolute reduction) in HbA1c from baseline value of 8.02% to 7.12% at 12-month follow-up. A significant reduction in fasting and post-prandial plasma glucose level of 25.47 and 108.44 mg/dL respectively from a mean baseline of 160.52 and 269.62 mg/dL, respectively (Tab. 2). Saroglitazar administration did not lead to weight gain. No serious adverse events were reported by the patients (Tab. 3).

Table 1. Demographic Parameters and Pattern of Glucose Lowering Drugs and Statin Therapy at Baseline (n = 150)

Parameter	Baseline value
Age [years]	57.6 ± 8.1
Male (%)	92 (61.3%)
Weight [kg]	71.3 ± 10.35
BMI	26.8 ± 3.89
Duration of diabetes [years]	7.8 ± 2.4
Smokers (%)	22 (14.6%)
Alcoholic (%)	4 (2.6%)
Single antidiabetic drug	8 (5.3%)
Dual antidiabetic therapy	47 (31.3%)
More than two antidiabetic drugs	95 (63.3%)
Atorvastatin	89 (59.4%)
Rosuvastatin	61 (40.7%)
Metformin	145 (96.6%)
Sulfonylurea	91 (60.6%)
DPP4 inhibitor	68 (45.3%)
SGLT2 inhibitor	13 (8.6%)
Alpha glucosidase inhibitors	23 (15.3%)
Insulin	18 (12%)
Hydroxychloroquine	11 (7.3%)
GLP1 agonist	2 (1.3%)

BMI — body mass index; DPP4 — dipeptidyl peptidase-4; GLP1 — glucagon-like peptide-1; SGLT2 — sodium-glucose cotransporter-2

Table 2. Changes in Lipid Parameters and HbA1c Level

Parameter	Baseline (week 0)	12th week	P	52nd week	P
Triglycerides [mg/dL]	669.93 ± 81.22	268.72 ± 82.32	0.0001	221.51 ± 61.81	0.001
Total cholesterol [mg/dL]	310.2 ± 33.04	240.7 ± 23.41	0.001	171.1 ± 21.82	0.001
Non-HDL-C [mg/dL]	270.8 ± 34.08	198.6 ± 28.02	0.001	128.3 ± 27.69	0.001
LDL-C [mg/dL]	167.68 ± 10.881	118.88 ± 12.16	0.001	103.17 ± 5.51	0.001
HDL-C [mg/dL]	40.42 ± 5.87	41.16 ± 6.13	0.059	42.33 ± 5.79	0.02
HbA1c [%]	8.02 ± 0.3	7.71 ± 0.5	0.001	7.12 ± 0.2	0.001
FPG [mg/dL]	160.52 ± 7.23	132.47 ± 5.81	0.001	119.62 ± 4.11	0.001
PPG [mg/dL]	269.62 ± 24.39	174.16 ± 16.31	0.001	161.18 ± 16.91	0.001
SGOT [U/L]	42.15 ± 3.18	41.14 ± 3.5	0.21	40.11 ± 3.4	0.31
SGPT [U/L]	37.34 ± 4.5	37.34 ± 4.5	0.23	36.32 ± 4.7	0.21
Serum creatinine [mg/dL]	0.7 ± 0.24	0.7 ± 0.22	0.53	0.7 ± 0.19	0.51
CPK [U/L]	76.3 ± 19.4	71.8 ± 24.2	0.62	65.3 ± 18.1	0.51

CPK — creatine phosphokinase; FPG — fasting plasma glucose; HDL-C — high-density lipoprotein cholesterol; HbA1c — glycated hemoglobin; LDL-C — low-density lipoprotein cholesterol; PPG — post prandial glucose; SGOT — serum glutamic oxaloacetic transaminase; SGPT — serum glutamic pyruvic transaminase

Table 3. Total Number of Adverse Events (AE) at the End of the Study

Type of AE	Visit 1 (12th week)	Visit 2 (52nd week)
Overall	15 (10%)	18 (12%)
Asthenia	2 (1%)	1 (0%)
Muscle pain	11 (7%)	8 (5%)
Dizziness	0	1 (0%)
Gastrointestinal	12 (8%)	9 (6%)
Hypoglycemia	2 (1%)	9 (6%)
Weight gain	1 (0%)	2 (1%)

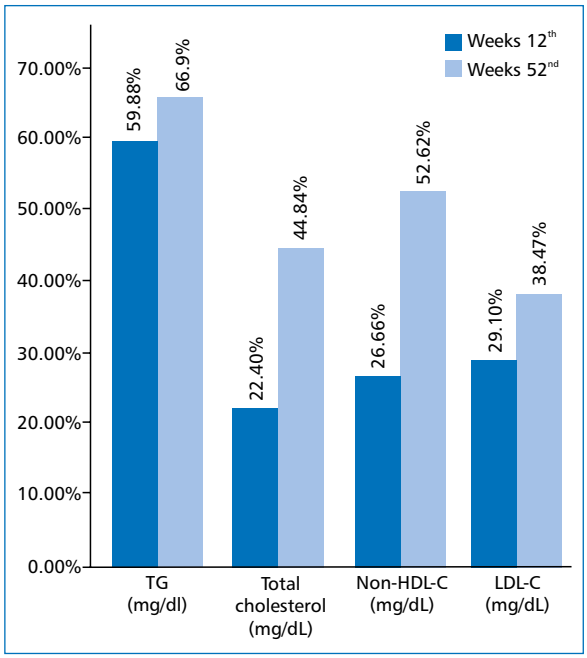

Figure 1. Reduction in Serum Lipid and Glycemic Parameters from Baseline
LDL-C — low-density lipoprotein cholesterol; non-HDL-C — non high-density lipoprotein cholesterol; TG — triglycerides

Discussion

The present study highlights the added benefit of saroglitazar when combined with statin and clearly points out that this combination has significant and marked advantage in improving all the lipid parameters. A statistically significant reduction in total cholesterol of 44.84 %, triglycerides of 66.9%, non-HDL-C of 52.62% and LDL-C of 38.47% was observed at the end of 52 weeks. When added to existing baseline antidiabetic medications, a statistical significant improvement in glycemic parameters was also observed, including 0.9% absolute reduction in HbA1c and improvement in both fasting and post prandial plasma glucose. In this study no serious adverse event which required medical attention were observed. There were no weight gain or edema and not any alteration in liver enzymes or serum creatinine were observed at the end of the study.

An associated low HDL-C and a higher triglycerides were observed in Indians as compared to Caucasians [13, 14]. Among major risk factors of cardiovascular disease (CVD), higher triglycerides were always considered as a strong one and recent advances has clearly mentioned that increase in CVD diseases were directly associated with higher TG levels. In a recent observational study of 34 years with more than 75,000 subjects from general population, it was observed that lower TG level was associated with lower CV risk. The group with TG < 90 mg/dL had 60% lower risk (statistically significant) of ischemic heart diseases than those with TG ≥ 360 mg/dL [15]. Another meta-analysis published in 2014 revealed that non-fasting TG of 600 mg/dL versus 72 mg/dL was associated with hazard ratio of 5.1 (95% CI 3.5−7.2) for myocardial infarction, 3.2 (2.5−4.1) for ischemic heart disease, 3.2 (2.2−4.7) for ischemic stroke, and 2.2 (1.8−2.7) for all-cause mortality [16]. Thus, higher TG level is found to be associated with increased cardiovascular risk. Although a few patients can develop pancreatitis with triglyceride levels > 500 mg/dL, the risk for pancreatitis is clinically significant when triglyceride levels are > 1000 mg/dL [6−8].

For the management of dyslipidemia, as per the 2020 American Diabetic Association guidelines, statin is recommended as primary therapy [17]. Even after statin therapy residual cardiovascular risk remains one of the major concern and it is postulated that atherogenic diabetic dyslipidemia is a major factor. A novel molecule, saroglitazar, is a dual PPAR α/γ agonist, approved for the management of DD in India.

In a recently conducted 24-week study with saroglitazar, researchers observed a significant decrease in non-HDL-C, sd-LDL-C, HbA1c from baseline: 142.3 ± ± 59.3 mg/dL to 109.9 ± 45.5 mg/dL; 32.5 ± 11.3 mg/dL to 25.9 ± 11.8 mg/dL and 8.1 ± 1.7 (%) to 6.9 ± 0.7 (%) and a significant increase in HDL-C from baseline: 37.3 ± ± 18.4 mg/dL to 43.4 ± 15.6 mg/dL [18]. In another large observational study of saroglitazar in 2804 patients with diabetic dyslipidemia, researchers observed a significant decrease in TG from 312.3 ± 122.7 mg/dL to 188.7 ± 61.4 mg/dL; non-HDL-C from 201.8 ± ± 64.1 mg/dL to149.4 ± 41.0 mg/ dL; HbA1c from 8.3 ± ± 1.3 (%) to 7.4 ± 0.9 (%); and significant increase in HDL-C from 38.8 ± 8.7 mg/dL to 41.0 ± 7.1 mg/dL after 12 weeks [12].

In a post marketing surveillance study of saroglitazar in 18 patients with type 2 diabetes with severe hypertriglyceridemia (baseline TG ≥ 1000 mg/dL), authors has ob-

served a significant decrease in TG (baseline: 1265.9 ± ± 394.3 mg/dL to week-12: 402.0 ± 221.8 mg/dL), non-HDL-C (baseline: 320.8 ± 172.8 mg/dL to week-12: 176.4 ± 62.9 mg/dL), and HbA1c [baseline: 8.9 ± 1.7 (%) to week-12: 7.8 ± 0.9 (%)] [19]. In a 58-week observational study of saroglitazar in 158 patients with diabetic dyslipidemia (baseline TG ≥ 150 mg/dL), authors found significant reduction in TG (baseline: 319.9 ± 178.8 mg/dL to week-58: 174.0 ± ± 113.6 mg/dL), non-HDL-C (baseline: 140.1 ± 55.4 mg/dL to week-58: 104.5 ± 49.7 mg/dL), and HbA1c [baseline: 7.9 ± 1.5 (%) to week-58: 7.3 ± 1.4 (%)] [20].

Limitations

This is a retrospective observational study, so medication compliance cannot be ascertained. The specific assigned laboratory was not used to conduct the pathological test. The sample size was small.

Conclusions

A significant improvement was observed in both glycemic and lipid parameters in patients with diabetic dyslipidemia having very high triglycerides (> 500 mg/dL) who were treated with saroglitazar 4 mg once daily for 52 weeks. Saroglitazar was well tolerated and no serious adverse event was reported throughout the study period. Randomized, controlled clinical trials with longer duration of follow up in this group of patients are needed.

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Conflict of interest

None declared.

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