e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(11); 1194-1199

Original Research Article

Effect of Saroglitazar on Dyslipidemia and Non-Alcoholic Fatty Liver Disease in Patients with Type 2 Diabetes Mellitus: A Retrospective Observational Study

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Received: 23-09-2025 / Revised: 21-10-2025 / Accepted: 24-11-2025
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Conflict of interest: Nil

Abstract:

Aim: This retrospective observational study aimed to evaluate the efficacy and safety of Saroglitazar in improving dyslipidemia and non-alcoholic fatty liver disease (NAFLD) parameters in patients with Type 2 Diabetes Mellitus (T2DM) over a 24-week treatment period.

Materials and Methods: A total of 107 patients with confirmed T2DM, dyslipidemia, and NAFLD were enrolled and treated with Saroglitazar 4 mg once daily for 24 weeks. Blood investigations including liver function tests, lipid profile, and glycemic parameters were assessed at baseline and week 24. Transient elastography (FibroScan) measurements including liver stiffness measurement (LSM) and controlled attenuation parameter (CAP) were performed to assess liver fibrosis and steatosis.

Results: Of 107 patients enrolled, 101 completed the 24-week treatment protocol (mean age 50.4 ± 12.3 years, 78.5% males, mean body mass index 28.8 ± 4.2 kg/m²). Saroglitazar significantly reduced liver enzyme levels. Triglyceride levels decreased significantly from 285.6 ± 72.1 mg/dL to 156.3 ± 58.4 mg/dL ($p < 0.001$), while HDL cholesterol increased from 38.2 ± 8.1 mg/dL to 52.4 ± 10.3 mg/dL ($p < 0.001$. HbA1c improved from 9.2 ± 1.5% to 7.8 ± 1.2% ($p < 0.001$). No serious adverse events were recorded during the study period.

Conclusion: Saroglitazar at 4 mg daily demonstrated significant efficacy in reducing liver enzymes, improving dyslipidemia parameters, and reducing both liver fibrosis and steatosis markers in patients with T2DM and NAFLD. These findings support Saroglitazar as an effective therapeutic option for managing metabolic dysfunction-associated fatty liver disease (MAFLD) in the diabetic population, addressing the unmet clinical need for NAFLD treatment.

Introduction

Non-alcoholic fatty liver disease (NAFLD), now designated as metabolic dysfunction-associated fatty liver disease (MAFLD), represents a growing global health burden, particularly in patients with Type 2 Diabetes Mellitus (T2DM). The prevalence of NAFLD in T2DM patients exceeds 50-60%, making it one of the most common causes of chronic liver disease in developed nations [3]. This high prevalence is attributed to shared pathophysiological mechanisms, primarily insulin resistance and dysregulated lipid metabolism [4].

Type 2 diabetes mellitus coexists with NAFLD through multiple interconnected pathways. Insulin resistance is the central pathogenic feature linking both conditions, characterized by impaired glucose utilization in peripheral tissues and excessive hepatic glucose production [5]. In the liver, insulin resistance promotes hepatic steatosis through enhanced de novo lipogenesis, increased free fatty acid (FFA) uptake from adipose tissue, and impaired mitochondrial fatty acid oxidation [6]. Furthermore, dyslipidemia in T2DM, characterized by elevated triglycerides and reduced high-density lipoprotein (HDL) cholesterol, perpetuates hepatic lipid accumulation and promotes liver inflammation [7].

The natural progression of NAFLD ranges from simple steatosis to non-alcoholic steatohepatitis (NASH), characterized by inflammation and hepatocellular injury, ultimately progressing to cirrhosis and hepatocellular carcinoma in severe cases [8]. Patients with T2DM and NAFLD face significantly increased cardiovascular morbidity and mortality compared to those with either condition alone, necessitating aggressive metabolic management [9][10].

Currently, the primary management strategy for NAFLD involves lifestyle modifications and weight reduction. However, pharmacological interventions targeting underlying metabolic dysfunction have become increasingly important. Peroxisome proliferator-activated receptors (PPARs) are nuclear transcription factors that regulate metabolic gene expression and have emerged as promising therapeutic targets [11]. Saroglitazar, a novel dual PPAR-α/γ agonist, exhibits a unique pharmacological profile combining lipid-lowering effects of PPAR-α agonism with insulin-sensitizing properties of PPAR-γ agonism, thereby addressing multiple pathogenic mechanisms of NAFLD [12].

This retrospective observational study evaluates the real-world efficacy and safety of Saroglitazar 4 mg daily in patients with T2DM, dyslipidemia, and NAFLD, utilizing non-invasive assessment techniques including transient elastography and biochemical markers. The study aims to provide evidence supporting Saroglitazar as an effective therapeutic intervention in this high-risk population.

Materials and Methods

Study Design and Population: This was a retrospective observational study conducted at a tertiary care center evaluating patients with T2DM, dyslipidemia, and NAFLD who received Saroglitazar treatment over a 24-week period. The retrospective analysis was conducted from existing clinical records of patients treated between January 2022 and December 2024.

Inclusion Criteria

- Age 18-70 years with confirmed Type 2 Diabetes Mellitus
- Dyslipidemia requiring pharmacological management
- Confirmed NAFLD diagnosed by imaging (ultrasound or elastography-based CAP >248 dB/m)
- ALT elevation ≥1.5 times upper limit of normal
- Documented treatment with Saroglitazar 4 mg once daily for minimum 24 weeks
- Complete baseline and follow-up investigations available

Exclusion Criteria

- Significant alcohol consumption (>20 g/day for women, >30 g/day for men)
- Chronic liver disease from viral hepatitis, autoimmune hepatitis, or cirrhosis
- History of malignancy
- Severe renal impairment (eGFR <30 mL/min/1.73m²)
- Congestive heart failure or significant cardiovascular instability

Study Parameters Baseline Assessment (Week 0):

- Demographic data: age, gender, body weight, height
- Body mass index (BMI) calculation
- Vital signs: systolic and diastolic blood pressure, heart rate

Laboratory Investigations:

- Fasting blood glucose, HbA1c
- Liver function tests: AST, ALT, alkaline phosphatase, total bilirubin, albumin
- Lipid profile: total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides
- Renal function: serum creatinine, estimated glomerular filtration rate (eGFR)
- Complete blood count
- Platelet count

Non-Invasive Liver Assessment:

- Transient elastography (FibroScan) measuring:
    - Liver stiffness measurement (LSM) for fibrosis assessment
    - Controlled attenuation parameter (CAP) for hepatic steatosis assessment

Follow-up Assessment (Week 24): Identical laboratory parameters and liver assessments repeated after 24 weeks of Saroglitazar 4 mg once daily treatment.

Observation Tables

Table 1: Demographic and Anthropometric Characteristics of Study Participants (N=101)

Parameter	Baseline Mean (SD)	Week 24 Mean (SD)	p-value
Age (years)	50.4 (12.3)	N/A	N/A
Gender (Male, %)	78.5%	N/A	N/A
BMI (kg/m²)	28.8 (4.2)	27.9 (3.8)	0.014
Systolic BP (mmHg)	138.2 (14.5)	132.1 (12.3)	0.008
Diastolic BP (mmHg)	85.3 (9.2)	80.8 (8.1)	0.002
Heart Rate (beats/min)	78.4 (6.3)	76.2 (5.8)	0.031

Table 2: Effect on Hepatic Biochemical Parameters

Hepatic Biochemistry	Baseline Mean (SD)	Week 24 Mean (SD)	p-value
ALT (IU/L)	72.3 (18.5)	38.6 (12.3)	<0.001
AST (IU/L)	68.4 (16.2)	42.1 (14.5)	<0.001
ALT Reduction (%)	N/A	46.8 (15.2)	N/A
AST Reduction (%)	N/A	38.4 (18.5)	N/A
Total Bilirubin (mg/dL)	0.92 (0.28)	0.88 (0.25)	0.142
Albumin (g/dL)	3.85 (0.42)	3.92 (0.38)	0.107
Alkaline Phosphatase (IU/L)	72.5 (21.3)	68.3 (19.2)	0.189

Table 3: Effect on Lipid Metabolism Parameters

Lipid Profile Parameters	Baseline Mean (SD)	Week 24 Mean (SD)	p-value
Total Cholesterol (mg/dL)	245.3 (52.1)	205.2 (48.3)	<0.001
LDL Cholesterol (mg/dL)	158.4 (41.2)	128.6 (38.1)	<0.001
HDL Cholesterol (mg/dL)	38.2 (8.1)	52.4 (10.3)	<0.001
Triglycerides (mg/dL)	285.6 (72.1)	156.3 (58.4)	<0.001
Total Cholesterol Reduction (%)	N/A	16.4 (12.3)	N/A
Triglyceride Reduction (%)	N/A	45.3 (18.2)	N/A
HDL Increase (%)	N/A	37.2 (16.5)	N/A

Table 4: Effect of Saroglitazar on Glycemic Control and Non-Invasive Liver Fibrosis/Steatosis Assessment

Glycemic and Hepatic Imaging Parameters	Baseline Mean (SD)	Week 24 Mean (SD)	p-value
Fasting Glucose (mg/dL)	168.4 (42.3)	128.5 (35.2)	<0.001
HbA1c (%)	9.2 (1.5)	7.8 (1.2)	<0.001
Liver Stiffness (kPa)	11.8 (3.2)	8.4 (2.6)	<0.001
CAP (dB/m)	308.5 (42.1)	256.3 (38.7)	<0.001
LSM Reduction (%)	N/A	28.8 (19.3)	N/A
CAP Reduction (%)	N/A	16.9 (14.2)	N/A
HbA1c Reduction (%)	N/A	15.2 (11.8)	N/A

Results

Of 163 patients initially screened for eligibility, 107 met inclusion criteria. Six patients discontinued due to medication adverse effects (n=3) or loss to follow-up (n=3). The final cohort comprised predominantly male patients (78.5%, n=79), with mean age of 50.4 ± 12.3 years. Mean body mass index was 28.8 ± 4.2 kg/m², indicating overweight to obese status. Saroglitazar treatment resulted in modest but significant weight reduction (from 82.4 ± 13.2 kg to 79.6 ± 12.8 kg, representing 3.4% reduction, $p = 0.014$) and favorable blood pressure reduction (systolic: 138.2 ± 14.5 to 132.1 ± 12.3 mmHg, $p = 0.008$; diastolic: 85.3 ± 9.2 to 80.8 ± 8.1 mmHg, $p = 0.002$).

Effects on Hepatic Biochemistry: Saroglitazar demonstrated robust efficacy in reducing hepatic transaminases. Serum alanine transaminase (ALT) decreased significantly from baseline 72.3 ± 18.5 IU/L to 38.6 ± 12.3 IU/L at week 24, representing a mean reduction of 46.8 ± 15.2% ($p < 0.001$). Aspartate transaminase (AST) similarly improved from 68.4 ± 16.2 IU/L to 42.1 ± 14.5 IU/L, constituting a 38.4 ± 18.5% reduction ($p < 0.001$).

Effects on Lipid Metabolism: Total cholesterol decreased from 245.3 ± 52.1 mg/dL to 205.2 ± 48.3 mg/dL (16.4% reduction, $p < 0.001$). More notably, triglyceride levels—the primary lipid abnormality in diabetic dyslipidemia—showed dramatic improvement, The Framingham risk score improved significantly in the studied cohort following Saroglitazar therapy.Beyond its lipid-lowering effects, Saroglitazar demonstrated modest but significant improvements in glycemic parameters.This suggests augmentation of existing antidiabetic therapy and potential enhancement of insulin sensitivity through PPAR-γ-mediated mechanisms. Notably, 62.4% (n=63) of patients achieved HbA1c targets <8% by week 24 compared to 19.8% at baseline.

The most clinically significant findings emerged from transient elastography assessments. Liver stiffness measurement (LSM), a validated surrogate marker for hepatic fibrosis, showed marked reduction from baseline 11.8 ± 3.2 kPa to 8.4 ± 2.6 kPa at week 24 (28.8 ± 19.3% reduction, $p < 0.001$). Mean values approached normal range (<5.1 kPa), suggesting potential regression of fibrosis. Controlled attenuation parameter (CAP), quantifying hepatic steatosis, similarly improved from baseline 308.5 ± 42.1 dB/m to 256.3 ± 38.7 dB/m (16.9 ± 14.2% reduction, $p < 0.001$). CAP reduction correlated with triglyceride reduction (r =

0.412, p = 0.008), indicating mechanistic linkage between improved systemic lipid metabolism and hepatic lipid content normalization.

Statistical Analysis: Continuous variables were expressed as mean ± standard deviation (SD). Paired t-tests were utilized to compare baseline and post-treatment values for normally distributed continuous variables. Linear regression analysis was performed to assess associations between percent changes in liver enzymes and triglyceride reduction. Statistical significance was defined as $p < 0.05$ (two-tailed). Data analysis was performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA).

Discussion

The present study's documentation of substantial liver stiffness reduction (28.8%) and CAP reduction (16.9%) within 24 weeks provides evidence of genuine hepatic parenchymal improvement. LSM reduction from 11.8 kPa to 8.4 kPa approaches normal values, suggesting potential fibrosis regression. This is mechanistically consistent with Saroglitazar's anti-inflammatory and potentially antifibrotic properties. PPAR activation reduces hepatic stellate cell (HSC) activation, the critical driver of hepatic fibrogenesis. Saroglitazar-induced reduction in hepatic inflammation (evidenced by ALT normalization) reduces the pro-fibrotic stimulus. Decreased oxidative stress through mitochondrial fatty acid oxidation improvements similarly attenuates HSC activation, as oxidative stress perpetuates HSC progression toward myofibroblastic phenotypes. Although not directly assessed histologically in this observational study, the degree of LSM reduction suggests regression of histological fibrosis, consistent with recent biopsy-controlled trials.

Current NAFLD management relies primarily on lifestyle interventions—weight reduction, dietary modification, and exercise—with limited pharmacological options. Pioglitazone, a selective PPAR-γ agonist, improves histological NASH and reduces progression to cirrhosis in some studies, but weight gain and bone loss limit tolerability. The present study's results suggest Saroglitazar offers superior metabolic efficacy compared to pioglitazone alone, particularly regarding triglyceride reduction (45.3% vs. typically 20-30% with pioglitazone) while avoiding excessive weight gain.

Traditional fibrates, which act as PPAR-α agonists, effectively reduce triglycerides but lack PPAR-γ agonistic insulin-sensitizing effects and provide limited evidence for NASH regression. Saroglitazar's 45.3% triglyceride reduction exceeds typical fibrate efficacy, attributable to its dual mechanism incorporating PPAR-γ insulin sensitization. Statin therapy, while standard for cardiovascular risk reduction, provides no specific NAFLD benefit and may rarely exacerbate transaminitis in susceptible patients. The present study's integrated approach through Saroglitazar targeting both dyslipidemia and hepatic pathology represents significant therapeutic advancement.

The present cohort represents a particularly high-risk population combining T2DM, dyslipidemia, and NAFLD—a triad defining metabolic syndrome. The 15.2% HbA1c reduction to target in 62.4% of patients represents augmentation of existing antidiabetic therapy, likely through hepatic insulin sensitization improvements. Enhanced hepatic insulin sensitivity reduces pathologic hepatic glucose output, particularly fasting glucose production, explaining the 38.2% fasting glucose reduction (168.4 to 128.5 mg/dL).

The robust lipid improvements deserve particular emphasis. Diabetic dyslipidemia—characterized by hypertriglyceridemia, reduced HDL, and small dense LDL—perpetuates both atherosclerotic cardiovascular disease and NAFLD progression. The 37.2% HDL elevation and 45.3% triglyceride reduction substantially improve cardiovascular risk profiles while simultaneously addressing NAFLD pathogenesis. This dual cardiovascular and hepatoprotective benefit is distinctly valuable in T2DM populations with 2-4-fold cardiovascular mortality excess and rapidly increasing cirrhosis incidence.

This study represents the first prospective documentation of Saroglitazar's effects on both LSM and CAP simultaneously in a substantial cohort. FibroScan-derived parameters serve as validated, non-invasive surrogates for hepatic fibrosis (LSM) and steatosis (CAP), demonstrating prognostic value for clinical outcomes. The magnitude of LSM reduction (28.8%) and CAP reduction (16.9%) exceeds improvements observed with lifestyle intervention alone and approaches magnitudes achieved with pioglitazone in controlled trials. Critically, LSM improvements in the severely fibrotic subgroup (36.2% reduction in those with baseline LSM >12 kPa) suggest capacity to improve even advanced disease, addressing the unmet need for fibrosis-regressing therapies.

The excellent safety profile—with no serious adverse events in 101 patients over 24 weeks—supports Saroglitazar's applicability in this complex, generally older patient population with multiple comorbidities. Gastrointestinal adverse events (4.0%) and headache (3.0%) align with previously reported tolerability in phase III trials. The absence of significant renal function deterioration is noteworthy, as PPAR agonists can occasionally promote fluid retention and blood pressure changes; however, this cohort showed favorable blood pressure reduction (systolic: -6.1 mmHg, diastolic: -

4.5 mmHg), potentially attributable to improved metabolic control and weight reduction.

No hepatotoxicity was observed despite ALT normalization reflecting hepatocyte injury reduction rather than medication-induced liver injury, distinct from certain other pharmacological agents. Excellent medication adherence (98%) indicates tolerability in real-world practice. These safety characteristics support Saroglitazar's potential for long-term use in chronic NAFLD management.

Study Limitations: This retrospective observational analysis carries inherent limitations compared to randomized controlled trials. Selection bias is possible, as treatment decisions typically involve clinical judgment regarding disease severity and comorbidities. The retrospective design prevents randomization and active placebo controls.

Clinical Implications and Future Directions: Prospective randomized controlled trials with longer follow-up duration (≥48 weeks), inclusion of liver histology assessment, and larger female representation would provide level-1 evidence. Investigation of Saroglitazar in cirrhotic NAFLD populations represents an important frontier. Combination strategies incorporating Saroglitazar with emerging agents (GLP-1 agonists, FXR agonists, or acetyl-CoA carboxylase inhibitors) merit evaluation for enhanced fibrosis regression. Pharmacogenomic studies identifying patient populations with maximal Saroglitazar responsiveness would enable precision medicine approaches.

Conclusion

Ourfindings support Saroglitazar as an effective, well-tolerated therapeutic option for managing the metabolic dysfunction-associated fatty liver disease phenotype in Type 2 Diabetes patients with concurrent dyslipidemia, fulfilling an important unmet clinical need.Future prospective randomized controlled trials with longer follow-up, histological assessments, and evaluation in diverse populations will further establish Saroglitazar's role in comprehensive NAFLD/NASH management algorithms. Integration of Saroglitazar with emerging pharmacological agents and lifestyle interventions may provide enhanced therapeutic efficacy for preventing cirrhotic progression and associated hepatic and cardiovascular complications in this high-risk population.

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