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Efficacy and Safety of Saroglitazar in Non-Alcoholic Fatty Liver Disease

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Abstract: ***Background:*** *Non-alcoholic fatty liver disease (NAFLD) is a leading cause of chronic liver disease and is associated with metabolic dysfunction, which can progress to liver fibrosis and cirrhosis. Currently, there are limited pharmacological options for the management of NAFLD. Saroglitazar, a dual PPARα/γ agonist, has shown promise in improving metabolic parameters and liver function.*
Aim of the study: *The study aims is to evaluate the efficacy and safety of saroglitazar in the management of NAFLD.*
Methods: *This prospective, open-label study was conducted on 162 adult patients diagnosed with NAFLD. Participants received saroglitazar 4 mg/day for 24 weeks. Baseline and follow-up assessments (at 12 and 24 weeks) were performed, including liver enzymes (ALT, AST), lipid profile (LDL, HDL, triglycerides), and liver stiffness measurement (kPa). The safety profile was assessed through the reporting of adverse events.*
Result: *Saroglitazar treatment led to significant improvements in liver function tests (ALT: 67.8±10.1 to 34.6±7.9 U/L, p<0.001; AST: 56.4±8.5 to 29.8±6.8 U/L, p<0.001), lipid profile (LDL: 152.5±18.4 to 118.6±14.7 mg/dL, p<0.001; triglycerides: 245.3±27.9 to 155.7±21.3 mg/dL, p<0.001; HDL: 38.5±4.2 to 45.1±4.5 mg/dL, p<0.001), and liver stiffness (11.2±1.8 to 8.5±1.3 kPa, p<0.001). A majority of patients (90.12%) showed improvement in ALT/AST levels, 85.19% exhibited lipid profile improvement, and 80.25% demonstrated a reduction in liver stiffness.*
Conclusion: *Saroglitazar demonstrated significant efficacy in improving liver function, lipid profile, and liver stiffness in patients with NAFLD. The drug was well-tolerated, with a favorable safety profile. Our findings support the potential of saroglitazar as an effective therapeutic option for managing NAFLD, warranting further large-scale studies for long-term validation..*
Keywords: *Saroglitazar, Non-Alcoholic Fatty Liver Disease, Pparα/Γ Agonist, Liver Enzymes, Lipid Profile, Liver Stiffness, Safety, Efficacy*

Original Research Article

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is a global health burden and a leading cause of chronic liver disease, closely linked to the rising prevalence of obesity and type 2 diabetes mellitus (T2DM) [1]. It encompasses a spectrum of liver conditions, from simple steatosis (fat accumulation in more than 5% of hepatocytes) to non-alcoholic steatohepatitis (NASH), which can advance to fibrosis, cirrhosis, and hepatocellular carcinoma [1]. NAFLD is recognized as the hepatic manifestation of metabolic syndrome, often characterized by insulin resistance, dyslipidemia, and a proinflammatory state

[2]. Its prevalence is approximately 30% in Asian populations and affects nearly one-third of the global population, with a higher incidence (70-80%) among individuals with Type 2 Diabetes Mellitus (T2DM) and obesity [2,3]. The pathogenesis of NAFLD involves lipid accumulation, insulin resistance, oxidative stress, and inflammation [4]. Lifestyle modifications, including exercise and weight loss, remain the cornerstone of NAFLD management, but these interventions are challenging to maintain long-term [5]. Furthermore, lifestyle changes alone are inadequate for advanced disease stages, highlighting the need for effective pharmacological treatments [6]. Despite significant progress in understanding NAFLD pathophysiology, the development of reliable therapies targeting key metabolic pathways has been limited [7]. Numerous drugs have been tested in the past decade, but none have achieved definitive therapeutic endpoints [7]. Peroxisome proliferator-activated receptors (PPARs) are nuclear receptors critical for regulating metabolic homeostasis, inflammation, cellular growth, and differentiation. PPARs exist in three main isoforms: alpha (α) in the liver, beta (β)/delta (δ) in skeletal muscle, and gamma (γ) in adipose tissue. Drugs targeting both PPARα and PPARγ, such as glitazars, have been explored for addressing dyslipidemia and insulin resistance (IR) in non-alcoholic fatty liver disease (NAFLD). However, the development of early glitazars (Tesaglitazar, Muraglitazar, Aleglitazar) was discontinued due to adverse effects linked to their significant PPARγ activity. Saroglitazar, a novel dual PPARα/γ agonist with predominant PPARα and moderate PPARγ activity, demonstrates a favorable safety profile, overcoming the limitations of earlier agents [8]. In South Asian countries like India, saroglitazar magnesium (Lipaglyn) is approved for type 2 diabetes (T2D) and dyslipidemia. It has shown promise in clinical trials for treating non-alcoholic steatohepatitis (NASH), highlighting its potential in managing metabolic syndrome-related disorders [9]. Unlike thiazolidinediones and fibric acid derivatives, saroglitazar is a unique compound that regulates lipid and glucose metabolism by activating PPARα and PPARγ. Its insulin-sensitizing properties stem from PPARγ activation, leading to a reduction in blood glucose levels. Concurrently, PPARα activation enhances hepatic fatty acid oxidation while suppressing triglyceride synthesis and secretion. This dual action promotes increased lipolysis and facilitates the clearance of triglyceride-rich lipoproteins from plasma by upregulating lipoprotein lipase (LPL) activity and downregulating Apo C-III, an endogenous inhibitor of LPL [10]. Additionally, saroglitazar lowers low-density lipoprotein (LDL) cholesterol levels, boosts the synthesis of apolipoproteins A-I and A-II, and elevates high-density lipoprotein (HDL) cholesterol. Currently approved for managing diabetic dyslipidemia in patients with inadequate response to statin therapy, saroglitazar holds promise for broader metabolic applications [10,11]. Despite these encouraging findings, there remains a need for further clinical evidence regarding the long-term safety and efficacy of saroglitazar, especially in comparison to other emerging pharmacologic agents. The study aims is to evaluate the efficacy and safety of saroglitazar in the management of NAFLD.

MATERIAL AND METHODS

This prospective, interventional, single-center observational study was designed to assess the efficacy and safety of saroglitazar in treating non-alcoholic fatty liver disease (NAFLD) at the Department of Hepatology in Dhaka Medical College and Hospital, Dhaka, Bangladesh. A total of 162 participants were screened for eligibility based on inclusion and exclusion criteria during one year from (Start) to (end). Finally, 162 patients were enrolled and analyzed in this study. Eligible participants provided informed consent before undergoing baseline evaluations, including physical examination, biochemical tests, and imaging studies. Participants received saroglitazar (4 mg) orally once daily for 24 weeks. Follow-up visits occurred during weeks 12 and 24 for monitoring and data collection. The ethical approval was taken from the ethics committee of the institution.

Inclusion Criteria:

- Adults aged 18 to 65 years were diagnosed with NAFLD via imaging or histological evidence.
- Elevated alanine aminotransferase (ALT) levels (≥45 U/L).
- Presence of metabolic syndrome components (e.g., central obesity, dyslipidemia, or insulin resistance).

Exclusion Criteria:

- Significant alcohol consumption (≥14 g/day for women and ≥28 g/day for men).
- Diagnosed cases of (e.g., viral hepatitis, autoimmune hepatitis, or hemochromatosis).
- History of malignancy or uncontrolled comorbidities.
- Pregnant or breastfeeding individuals.
- Use of medications known to influence liver enzymes within 3 months before enrollment.

Data Collection:

Baseline data, including age, gender, BMI, and comorbidities, were meticulously collected at the initial visit, with follow-up assessments conducted at weeks 12 and 24 to ensure comprehensive longitudinal analysis. Parameters evaluated included liver enzymes (ALT, AST) and lipid profiles (LDL, HDL, triglycerides) to monitor metabolic and hepatic function. Liver stiffness measurement (LSM) was performed using transient elastography for non-invasive assessment of hepatic fibrosis. Anthropometric measurements, such as BMI and waist circumference, were systematically recorded to

track physical changes. Additionally, adverse events were rigorously documented to assess safety and tolerability throughout the study period.

Parameters Assessed and Follow-Up

Participants in the 24-week intervention were meticulously monitored, with medication adherence tracked through patient diaries and pill counts. Primary efficacy endpoints focused on improvements in ALT levels, lipid profiles, and liver stiffness, while secondary endpoints included AST level changes and the frequency of adverse events. Biochemical parameters such as ALT, AST, LDL, HDL, and triglycerides were analyzed using standardized laboratory methods, and liver stiffness was measured via FibroScan. Adverse events were documented and graded for severity using the Common Terminology Criteria for Adverse Events (CTCAE).

Data Analysis:

Statistical analyses were performed using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation (SD) and compared using paired t-tests or ANOVA. Categorical variables were expressed as frequencies and percentages and analyzed using chi-square tests. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 162 participants were included in this study. The mean age was 42.8±8.6 years, and the average BMI was 29.5±3.2 kg/m². Males comprised 57.41% of the study population, while females accounted for 42.59%. Diabetes was the most common comorbidity, affecting 30.25% of participants, followed by hypertension in 25.31%, hypothyroidism in 19.75%, and coronary artery disease in 14.81% (Table 1). During the follow-up period, significant improvements were observed across all measured parameters. ALT levels showed a progressive decline from a baseline value of 67.8±10.1 U/L to 45.2±8.7 U/L at 12 weeks and further decreased to 34.6±7.9 U/L at 24 weeks. AST levels followed a similar trend, reducing from 56.4±8.5 U/L at baseline to 39.1±7.4 U/L at 12 weeks and 29.8±6.8 U/L at 24 weeks. Lipid profile parameters also demonstrated notable changes, with LDL levels decreasing from 152.5±18.4 mg/dL at baseline to 134.2±15.8 mg/dL at 12 weeks and 118.6±14.7 mg/dL at 24 weeks. Conversely, HDL levels improved over time, rising from 38.5±4.2 mg/dL at baseline to 42.3±4.7 mg/dL at 12 weeks and reaching 45.1±4.5 mg/dL at 24 weeks. Triglyceride levels showed a substantial reduction from 245.3±27.9 mg/dL at baseline to 196.4±24.6 mg/dL at 12 weeks and further declined to 155.7±21.3 mg/dL at 24 weeks. Liver stiffness measurements also indicated significant improvement, with values decreasing from 11.2±1.8 kPa at baseline to 9.7±1.5 kPa at 12 weeks and 8.5±1.3 kPa at 24 weeks. All observed changes were statistically significant (p<0.001) (Table 2). Table 3 showed the study outcome and adverse events. A majority of participants (90.12%) showed improvement in ALT and AST levels, while 85.19% exhibited enhancements in their lipid profiles. Additionally, liver stiffness was reduced in 80.25% of the study population, indicating positive treatment effects. Adverse events were reported in a small proportion of participants, with nausea and fatigue each affecting 4.94% of individuals, while diarrhea was observed in 2.47% of cases.

Table 1: Baseline characteristics of the study population (N=162)

Variable	Frequency (n)	Percentage (%)
	Mean ± SD	
Age (years)	42.8 ± 8.6	
BMI (kg/m²)	29.5 ± 3.2	
Gender		
Male	93	57.41
Female	69	42.59
Comorbidities		
Diabetics	49	30.25
Hypertension	41	25.31
Coronary Artery Disease	24	14.81
Hypothyroidism	32	19.75

Table 2: Comparison of parameter during follow up

Parameter	Baseline	Week 12	Week 24	p-value
ALT (U/L)	67.8 ± 10.1	45.2 ± 8.7	34.6 ± 7.9	<0.001
AST (U/L)	56.4 ± 8.5	39.1 ± 7.4	29.8 ± 6.8	<0.001
LDL (mg/dL)	152.5 ± 18.4	134.2 ± 15.8	118.6 ± 14.7	<0.001
HDL (mg/dL)	38.5 ± 4.2	42.3 ± 4.7	45.1 ± 4.5	<0.001
Triglycerides (mg/dL)	245.3 ± 27.9	196.4 ± 24.6	155.7 ± 21.3	<0.001
Liver Stiffness (kPa)	11.2 ± 1.8	9.7 ± 1.5	8.5 ± 1.3	<0.001

Table 3: Study outcome and adverse events.

Outcome	Frequency (n)	Percentage (%)
Improvement in ALT/AST	146	90.12
Improvement in Lipid Profile	138	85.19
Reduction in Liver Stiffness	130	80.25
Adverse Events Reported		
Nausea	8	4.94
Diarrhea	4	2.47
Fatigue	8	4.94

DISCUSSION

Non-alcoholic fatty liver disease (NAFLD) is a leading cause of chronic liver disease globally, with a rising prevalence due to the increasing burden of obesity, diabetes, and metabolic syndrome [12]. It encompasses a spectrum ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, and cirrhosis, ultimately increasing the risk of hepatocellular carcinoma and cardiovascular disease [13]. Currently, there are no approved pharmacological therapies for NAFLD, and treatment strategies primarily focus on lifestyle modifications and metabolic control. Given the complex pathophysiology of NAFLD, targeting both hepatic inflammation and dyslipidemia is crucial for effective management. Saroglitazar, a dual peroxisome proliferator-activated receptor (PPAR)-α/γ agonist, has shown potential in improving liver function and metabolic parameters, making it a promising therapeutic option [14]. In our study, Saroglitazar demonstrated significant efficacy in improving hepatic function, lipid parameters, and liver stiffness in patients with non-alcoholic fatty liver disease (NAFLD). The reduction in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels observed over the 24-week treatment period suggests a hepatoprotective effect, consistent with its role as a dual peroxisome proliferator-activated receptor (PPAR)-α/γ agonist. These findings are in concordance with previous reports indicating that Saroglitazar reduces hepatic inflammation and improves liver enzyme profiles in patients with NAFLD [15]. Furthermore, the present study demonstrated a significant improvement in lipid profile, with reductions in low-density lipoprotein (LDL) and triglyceride levels alongside an increase in high-density lipoprotein (HDL). This is particularly relevant given that dyslipidemia is a well-recognized pathogenic factor in NAFLD progression [16]. Previous studies have reported that Saroglitazar effectively lowers atherogenic lipid parameters, thereby addressing both hepatic and cardiovascular risks associated with NAFLD [17]. These findings further substantiate the metabolic benefits of Saroglitazar beyond liver function improvement. Another notable finding was the significant reduction in liver stiffness, as assessed by transient elastography, suggesting a potential antifibrotic effect. Devadas et al. (2022) reported similar findings, suggesting that saroglitazar may be a promising therapeutic option for reducing liver stiffness and transaminitis in patients with nonalcoholic fatty liver disease (NAFLD) [15]. Additionally, Padole (2022) demonstrated in his study that saroglitazar treatment resulted in a significant reduction in liver stiffness measurements (LSM), further supporting its potential in managing liver-related complications in NAFLD [18]. In prior studies, Saroglitazar has been associated with a decrease in hepatic fibrosis markers, reinforcing its role in ameliorating liver stiffness and fibrosis progression [19]. Given that fibrosis remains a critical determinant of NAFLD prognosis, these observations hold clinical significance. Despite the evident efficacy, the safety profile of Saroglitazar warrants consideration. Adverse events were reported in a small proportion of participants, with nausea (4.94%), diarrhea (2.47%), and fatigue (4.94%) being the most frequently observed. Notably, these adverse effects were mild and did not lead to treatment discontinuation. Similar tolerability has been reported in previous studies, suggesting that Saroglitazar is well tolerated in NAFLD patients [20].

Nevertheless, it is imperative to acknowledge certain limitations. Firstly, the sample size in our study was relatively small (N = 162), and there was no placebo control group. Consequently, while the results are promising, they should be interpreted with caution. Moreover, our study duration was limited to 24 weeks, which may not capture the long-term histological benefits or potential delayed adverse effects of Saroglitazar.

CONCLUSION AND RECOMMENDATIONS

The present study demonstrates that Saroglitazar is efficacious in improving key biochemical and imaging parameters in NAFLD patients, with a favorable safety profile. Moreover, the significant improvements observed in liver enzymes, lipid profiles, and liver stiffness reinforce the potential role of Saroglitazar as a therapeutic option for NAFLD. Consequently, future research should focus on confirming these benefits in larger, controlled studies and on elucidating the mechanisms underlying these improvements.

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