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EFFECT OF DUAL PPAR α/γ AGONIST SAROGLITAZAR ON LIPID PROFILE AND LIVER ENZYMES IN NAFLD PATIENTS

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Abstract

Background: The metabolic syndrome demonstrates as nonalcoholic fatty liver disease (NAFLD), which is characterized by hepatic fat accumulation linked to varying degrees of inflammation and fibrosis. The NAFLD constitutes a broad category of chronic liver disease, encompassing nonalcoholic fatty liver, nonalcoholic steatohepatitis, and cirrhosis.

Objective: The goal of the current investigation was assessing effect of dual PPAR α/γ agonist saroglitazar on lipid profile and liver enzymes in NAFLD patients

Material and method: After an overnight fast, all 240 study participants provided blood samples in accordance with the protocol for the measurement of serum lipids and lipoproteins. The lipid profile and liver enzymes parameters were assessed. The patients on continued saroglitazar 4 mg once daily therapy was follow-up at 24 and 52 weeks. SPSS version 20 was used for the statistical analysis, and $p<0.05$ was chosen as the significance level.

Result: Among 240 patients, there was significant increase in serum HDL – baseline 40.57 ±4.21mg/dl, at 24 weeks 43.97± 4.61 mg/dl and at 52 weeks 47.08± 4.94mg/dl. Over 52 weeks, HDL increased while LDL, triglycerides, and total cholesterol all declined significantly ($P < 0.001$). Such profound improvement in dyslipidemia—with up to ~40–45% TG reduction—is a hallmark of saroglitazar therapy in real-world cohorts and phase II/III data. At baseline, mean alanine aminotransferase (ALT) levels were 49.94 ± 20.04 U/L, which decreased significantly to 33.96 ± 13.64 U/L at 24 weeks and further to 31.95 ± 12.81 U/L at 52 weeks ($p < 0.05$). Similarly, aspartate aminotransferase (AST) showed a marked reduction from 58.73 ± 36.35 U/L at baseline to 42.28 ± 26.14 U/L at 24 weeks and 39.95 ± 24.70 U/L at 52 weeks ($p < 0.05$).

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Conclusion: These findings suggest that saroglitazar 4 mg daily may be a promising therapeutic option for non-diabetic NAFLD patients, providing multi-dimensional benefits across liver enzymes and lipid profile.

Keywords: NAFLD, liver enzymes, lipid profile, saroglitazar.

1. INTRODUCTION

The metabolic syndrome demonstrates as nonalcoholic fatty liver disease (NAFLD), which is characterized by hepatic fat accumulation linked to varying degrees of inflammation and fibrosis. The NAFLD constitutes a broad category of chronic liver disease, encompassing nonalcoholic fatty liver, nonalcoholic steatohepatitis, and cirrhosis. About one-third of the general population is impacted, and 70–75% of people with diabetes and obesity are linked to it [1].

It is typified by hepatic fat buildup in people who do not consume statistically significant amounts of alcohol, and it is specifically linked to metabolic syndrome includes obesity, dyslipidemia, insulin resistance, and hypertension. It has been demonstrated that NAFLD is both independently and substantially linked to an elevated risk of cardiovascular disease and type 2 diabetes mellitus (T2DM). [2]

Globally, the prevalence of NAFLD has increased exponentially; according to recent research, 25% of people worldwide suffer from the disease, and between 2015 and 2030, it is expected to climb by 63%[3,4] . According to reports, the incidence of adult NAFLD in India ranges from 6.7% to 55.1%. NAFLD may be the cause of almost one-third of all patients with an asymptomatic increase in liver enzymes. Additionally, liver transplant centers' explant histology data indicate that NAFLD was present in two-thirds of patients with "cryptogenic" cirrhosis. In India, among the healthy population, the prevalence of pediatric NAFLD ranges from 7.3% to 22.4%. As people age, the prevalence of NAFLD rises[5]. In contrast to the sharp increase in the prevalence of sickness, there hasn't been much advancement in the treatment arsenal against NAFLD[3]. Although liver-directed therapy is an option for individuals with non-alcoholic steatohepatitis (NASH) who have stage 2 or greater fibrosis, the only available therapeutic options are dietary and lifestyle changes, risk factor reduction, and, in certain situations, the use of vitamin E and pioglitazone[6] .

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There are few pharmacotherapeutic alternatives available for treating NAFLD, and lifestyle modifications have been the mainstay of treatment, which is challenging for the majority of patients to follow. The influence of pharmacological therapies on liver fibrosis should be the primary indicator of their effectiveness in treating NAFLD, since the degree of fibrosis has been connected to both hepatic and extrahepatic morbidity and death in NAFLD.[7]

Peroxisome proliferator-activated receptors (PPARs) are nuclear receptors that are essential for controlling inflammation, cellular proliferation, differentiation, and metabolic homeostasis. There are primarily three isoforms: gamma (γ) in adipose tissue, beta (β)/delta (δ) in skeletal muscle, and alpha (α) in the liver. The focus is on drugs that act on both PPARα and γ (glitazars), which address two significant problems of NAFLD: dyslipidemia and IR. The development of some glitazars, including Tesaglitazar, Muraglitazar, and Aleglitazar, was stopped because of negative side effects resulting from their strong γ activity. With a significant PPARα impact and a minor PPARγ effect, Saroglitazar is a new dual PPARα/γ agonist that has no adverse effects. In 2013, Saroglitazar was approved by the Drug Controller General of India to treat DD sufferers.[7]

A new non-invasive method for evaluating hepatic fibrosis and steatosis is vibration-controlled transient elastography, or FibroScan. Liver stiffness measurement (LSM) and controlled attenuation parameter (CAP), which are correlated with fibrosis and steatosis, respectively, are produced by delivering shear waves across the right lobe of the liver and measuring the returning shear wave velocities. The benefits of transient elastography (TE) are its speed, non-invasiveness, tolerance, and ability to cover 100 times the liver volume compared to liver biopsy. According to reports, TE is a precise and trustworthy non-invasive technique for evaluating liver fibrosis and steatosis in NAFLD patients[8–11].

2. MATERIAL AND METHOD

Study Site: The study was conducted in the Department of Pharmacology and Department of General Medicine of Index Medical College & Hospital and Research centre, Indore, M.P (a tertiary healthcare teaching hospital attached to Malwanchal University, Indore, M.P).

Study Design: A prospective, uni-centric, observational, hospital-based study. Total duration of study was 03 years.

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Study Population: A total of 240 diagnosed patients of Non-Alcoholic Fatty Liver Disease (NAFLD) and satisfying the inclusion and exclusion criteria mentioned below was enrolled in the study.

Inclusion Criteria:

1. Patients of both gender and aged from 18 to 60 years.

2. Documented diagnosis of NAFLD according to the American Association for the Study of Liver Diseases (AASLD) criteria.

3. Patients on lifestyle modification for NASH for at least one month.

4. Willingness to comply with all protocol required evaluations; provision of written informed consent before any study specific tests or procedures were performed.

5. Patients providing a written informed consent for participation in this study.

Exclusion criteria:

1. Pregnancy and lactation.

2. Known allergy, sensitivity, or intolerance to the study drug.

3. History of alcohol consumption of >30 gm/week for men and >20 gm/week for women for 3 consecutive months in the last 5 years.

4. Patient on vitamin E (>400 IU/day) or multivitamins containing vitamin E (>400 IU/day) or fibrates (clofibrate, fenofibrate) in the 3 months preceding enrolment.

5. Patient on drugs with potential effect on NAFLD such as ursodeoxycholic acid, S-adenosylmethionine (SAM-e), glutathione, Orlistat, Betaine, and Pentoxyphyllin 1 month prior to enrolment.

6. Use of drugs associated with a clinical or histological picture consistent with fatty liver diseases for more than 3 months in n the 1 year prior to start of the study, namely, Amiodarone, Tamoxifen, Methotrexate, Glucocorticoids, anabolic steroids, Tetracyclines, estrogens, Valproic acid, Chloroquine, anti-HIV drugs.

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7. History of other cause of chronic liver disease (viral hepatitis B or C, autoimmune hepatitis, cholestatic and metabolic liver diseases, and hemochromatosis.

8. Patients with known cirrhosis (compensated/decompensated) either based on clinical criteria or liver histology or Imaging techniques.

9. History of myopathies or evidence of active muscle disease

10.History of malignancy in the past 5 years and/or active neoplasm with the exception of resolved superficial non-melanoma skin cancer

11.History of bowel surgery (gastrointestinal (bariatric) or undergoing evaluation for bariatric surgery for obesity, extensive small-bowel resection, or orthotopic liver transplant (OLT) or listed for OLT.

12.History or other evidence of severe illness or any other conditions that would make the patient, in the opinion of the investigator, unsuitable for the study (such as poorly controlled psychiatric disease, HIV, coronary artery disease or active gastrointestinal conditions that might interfere with drug absorption)

Sample Size: For the 95% confidence interval, desired sample size was calculated using the formula: $n = Z2pq/e2$

Where,

n = Sample size needed

p = Prevalence rate from previous study q = (1-p)

Z = the value from the table of probabilities of the standard normal distribution for the desired

e = the margin of error

The estimated prevalence of NAFLD is around 9% to 32% in general population of India according to operational guidelines for NAFLD by NPCDCS. With 95% confidence level, considering 5% margin of error sample size comes out to be 251.Among these, all those who fulfil the inclusion criteria and consent to participate in the study was recruited for study intervention.

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Study Method: After an overnight fast, all study participants provided blood samples in accordance with the protocol for the measurement of serum lipids and lipoproteins. The lipid parameters assessed included total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), very-low-density lipoprotein cholesterol (VLDL-C), and non-HDL cholesterol. Liver function test was done to measure ALT and AST at baseline time. The patients on continued saroglitazar 4 mg once daily therapy was follow-up at 24 and 52 weeks. Anthropometric measurements were taken at each clinical visit. Patients were undergoing testing for lipid profile, LFT, HbA1c, and CBC at baseline, 24 weeks, and 52 weeks. The data was analyzed using paired t-test on SPSS Version 28.0 statistical package.

3. RESULTS

Total 240 patients were included in study. They had a mean age of 46.8 ± 10.05 years, predominantly male (69.2%), with a high prevalence of type 2 diabetes (69%) and less frequent hypertension (9.6%), which typically include middle-aged patients with metabolic comorbidities undergoing saroglitazar 4 mg therapy. Gender distribution showed males with 69.17% population while female formed 30.83% of study population.

	Baseline		**24 weeks**		**52 weeks**		**P value**
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	
High-Density Lipoprotein (mg/dL)	40.57	4.21	43.97	4.61	47.08	4.94	<0.001
Low-Density Lipoprotein (mg/dL)	126.22	28.93	106.02	24.33	94.65	21.72	<0.001
Triglycerides (ng/dL)	163.24	33.59	115.10	23.70	97.94	20.15	<0.001
Total Cholesterol (μL)	181.65	33.37	150.78	27.70	136.24	25.01	<0.001

Table 1: Lipid Profile

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The mean triglyceride level at the start of study was 163.24 mg/dl in the study. There was significant decrease in serum triglycerides – baseline 163.24 ± 33.59 mg/dl, at 24weeks 115.10± 23.70mg/dl and at 52weeks 97.94 ± 20.17 mg/dl respectively. The mean cholesterol at the start of study was 181.65 mg/dl. There was significant decrease in serum cholesterol – baseline 181.65 ±33.37mg/dl, at 24 weeks 150.78± 27.70 mg/dl and at 52 weeks 136.24± 25.01mg/dl. Similarly, there was significant decrease in serum LDL – There was significant decrease in serum cholesterol – baseline 126.22 ±28.93mg/dl, at 24 weeks 106.02± 24.33 mg/dl and at 52 weeks 94.65± 21.72mg/dl. There was significant increase in serum HDL – baseline 40.57 ±4.21mg/dl, at 24 weeks 43.97± 4.61 mg/dl and at 52 weeks 47.08± 4.94mg/dl. Over 52 weeks, HDL increased while LDL, triglycerides, and total cholesterol all declined significantly ($P < 0.001$). Such profound improvement in dyslipidemia—with up to ~40–45% TG reduction—is a hallmark of saroglitazar therapy in real-world cohorts and phase II/III data.

	Baseline		24 weeks		52 weeks		**P value**
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	
Alanine Aminotransferase	49.94	20.04	33.96	13.64	31.95	12.81	<0.05
Aspartate Aminotransferase	58.73	36.35	42.28	26.14	39.95	24.70	<0.05

Table 2: Liver Function Test (ALT & AST))

At baseline, mean alanine aminotransferase (ALT) levels were 49.94 ± 20.04 U/L, which decreased significantly to 33.96 ± 13.64 U/L at 24 weeks and further to 31.95 ± 12.81 U/L at 52 weeks ($p < 0.05$). Similarly, aspartate aminotransferase (AST) showed a marked reduction from 58.73 ± 36.35 U/L at baseline to 42.28 ± 26.14 U/L at 24 weeks and 39.95 ± 24.70 U/L at 52 weeks ($p < 0.05$). These results demonstrate a sustained and statistically significant improvement in liver enzyme levels over the study duration, highlighting the potential therapeutic benefit of the intervention in improving hepatic function among the study population.

4. DISCUSSION

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The current investigation evaluates the effectiveness of saroglitazar in non-diabetic NAFLD patients by comparing changes in liver enzymes and fibroscan parameters before and after treatment. All participants were administered saroglitazar 4 mg, as per existing recommendations and evidence [7,12]. To the best of our knowledge, this is the first study from India to highlight the therapeutic benefits of saroglitazar specifically in non-diabetic NAFLD cases. An ideal pharmacologic intervention for NAFLD should positively influence insulin sensitivity, hepatic fat accumulation, hepatocellular inflammation, oxidative stress, mitochondrial dysfunction, and liver fibrosis [13].

Previously, a variety of drugs were trialed for NAFLD management, though with limited success. One of the primary events in NAFLD pathogenesis is the intracellular fat accumulation in hepatocytes. PPARs play a vital role in lipid metabolism regulation. PPAR α is primarily found in hepatocytes and its activation prevents hepatic fat buildup and progression to steatohepatitis. Conversely, PPAR γ is mainly expressed in adipose tissue, and its stimulation enhances insulin sensitivity, thereby reducing the delivery of fatty acids to the liver [14]. Pioglitazone, a PPAR γ agonist, showed histological improvements due to its antifibrotic effects but was not approved due to adverse effects such as heart failure, vision problems, weight gain, and increased bladder cancer risk [15]. PPAR α agonists like fibrates failed to demonstrate clinical benefit in NAFLD trials [16]. Elafibrinor, a PPAR-α/δ agonist, is currently under evaluation for its potential in NAFLD treatment[17] . Older therapies like metformin, ursodeoxycholic acid, and vitamin E were previously used, but current research does not support their effectiveness[18,19] . More recently, encouraging findings were published about obeticholic acid in NAFLD, though more comprehensive data is needed to confirm its safety and efficacy before formal approval[16] . Saroglitazar, a dual PPAR α/γ agonist, has gained approval for NAFLD management. Jain N et al. observed that saroglitazar reduced dyslipidemia and improved insulin resistance by mitigating glucolipotoxicity and activating PPAR γ in diabetic patients with lipid disorders [20]. Elevated ALT and AST are considered indicators of liver cell inflammation and are useful for identifying NASH in the absence of histology. Saroglitazar effectively lowers these enzyme levels, indicating decreased hepatic inflammation. In this study, a notable reduction in liver enzymes was found after 24 weeks of therapy, aligning with prior studies. Kaul U et al. also reported decreased ALT levels following 12 to 58 weeks of saroglitazar treatment[12] . Another study in animal models found a

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60% drop in ALT and 43% in AST after 12 weeks of therapy [21]. Similarly, Goyal O et al. recorded substantial reductions in ALT and AST after 24 weeks of treatment [7]. Hence, the study concludes that 24-week treatment aids in resolving transaminitis and reflects reduced hepatic inflammation. Our findings revealed a significant decrease in LSM following saroglitazar treatment. Goyal O et al. also documented improvements in LSM after 24 weeks of therapy[7] .

5. CONCLUSION

These findings suggest that saroglitazar 4 mg daily may be a promising therapeutic option for non-diabetic NAFLD patients, providing multi-dimensional benefits across liver enzymes, steatosis and fibrosis markers, glycemic control, and lipid regulation. Although the study was observational and lacks a comparator arm, the improvements observed align closely with prior clinical and real-world evidence, supporting its potential role in NAFLD management. Further trials, ideally randomized and including histologic endpoints, would be invaluable to confirm these promising results.

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