Saroglitazar for Nonalcoholic Fatty Liver Disease: A Single Centre Experience in 91 Patients

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Background: **Saroglitazar is a novel, dual peroxisome proliferator-activated receptors-α/γ agonist and is being investigated for the treatment of nonalcoholic fatty liver disease (NAFLD).** ***Patients and methods:*** **Consecutive overweight (body mass index [BMI] >23 kg/m^2) patients of NAFLD, diagnosed based on controlled attenuation parameter (CAP) >248 dB/m, and attending the outpatient department of a tertiary care centre in New Delhi, were enrolled. Patients with cirrhosis (liver stiffness measurement [LSM] >13.5 kPa) and those with concomitant liver disease due to other aetiologies (alcohol, viral, etc.) were excluded. All patients received saroglitazar 4 mg/day; in addition, they were advised to reduce weight and were counselled regarding diet and exercise. At 3-month follow-up, patients were categorized into those who were able to reduce ≥5% body weight and those who could n'ot, and both these groups were compared.** ***Results:*** **A total of 91 patients (median age 45 years [range 18–66 years]; 81% men) were included in the study. The median BMI was 29.3 kg/m^2 (range 23.6–42.2 kg/m^2). The baseline median (range) aspartate transaminase, alanine transaminase, gamma glutamyl transferase, LSM and CAP values were 40 IU/dL (range 22–144 IU/dL), 48 IU/dL (range 13–164 IU/dL), 42 IU/dL (range 4–171 IU/dL), 6.7 kPa (range 3.6–13.1 kPa), and 308 dB/m (range 249–400 dB/m). All patients tolerated saroglitazar well. At 3-month, 57 patients (63%) were able to reduce ≥5% weight, whereas in the remaining 34 patients (37%), the weight reduction was <5% from baseline. Transaminases values improved in both the groups; however, LSM and CAP values improved only in patients who reduced weight.** ***Conclusion:*** **In overweight patients with NAFLD, a 3-month therapy with saroglitazar is able to improve transaminases but not LSM and CAP values unless accompanied by weight reduction of at least 5%. Larger randomized controlled trials are needed to document the independent effect of saroglitazar in these patients.** **(J Clin Exp Hepatol 2022;12:435–439)**

NAFLD

Nonalcoholic fatty liver disease (NAFLD) is a spectrum of disease ranging from bland fat accumulation (steatosis, nonalcoholic fatty liver) in more than 5% hepatocytes to, inflammation of the liver with or without fibrosis (nonalcoholic steatohepatitis [NASH]), to cirrhosis progressing to end-stage liver disease, and hepatocellular carcinoma.[1] Owing to the rising prevalence of obesity and other metabolic risk factors, NAFLD has emerged as the most common cause of end-stage liver disease and liver transplantation in many parts of the world.

Lifestyle modification and weight reduction in obese subjects remain the cornerstone of therapy for NAFLD as they improve liver histology, transaminitis and quality of life.[2,3] Early studies had suggested that weight loss of 7%–10% was associated with histologic improvement in steatosis and inflammation, whereas recent work has shown that as little as 5% weight loss may result in regression of fibrosis.[3–5] However, these weight loss goals are rarely achieved by lifestyle modifications alone. More importantly, lifestyle changes alone are insufficient to stop disease progression, especially for patients who are at later stages of the disease where there are ongoing liver inflammation and fibrosis. Owing to these reasons, the quest for effective drug for NAFLD continues.[6]

Pharmacological treatment of NAFLD remains elusive. In the last 12 years, after vitamin E[7] and pioglitazone,[7,8] seven other drugs have shown promise as a drug treatment for NASH in phase 2–3 trials: Obeticholic acid,[9] cenicriviroc,[10,11] lanifibranor,[12] resmetirom,[13] liraglutide,[14] saroglitazar[15,16] and the latest being semaglutide.[17] However, till now, none of these drugs have received FDA approval, and the mainstay of treatment of NASH, in the world over, remains lifestyle modifications, including regular physical exercise and consuming a hypocaloric diet.

Keywords: saroglitazar, pPAR agonist, NAFLD, obesity, controlled attenuation parameter

Received: 7.2.2021; *Accepted:* 12.6.2021; *Available online 19 June 2021*

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Abbreviations: ALP: alkaline phosphatase; ALT: alanine transaminase; AST: aspartate transaminase; BMI: body mass index; CAP: controlled attenuation parameter; dB: decibels; DCGI: Drug Controller General of India; FDA: Food and Drug Administration; GGT: gamma glutamyl transferase; HCV: hepatitis C virus; IQR: interquartile range; IU: international units; kPa: kilopascal; LSM: liver stiffness measurement; MAFLD: metabolic (dysfunction) associated fatty liver disease; NAFLD: nonalcoholic fatty liver disease; NASH: nonalcoholic steatohepatitis; PPAR: peroxisome proliferator-activated receptor

https://doi.org/10.1016/j.jceh.2021.06.015

Saroglitazar, a novel dual peroxisome proliferator-activated receptors (PPAR) alfa/gamma agonist used for diabetic dyslipidaemia,[18] targets some pathogenic mechanisms of NASH.[19] PPAR alfa agonism targets lipid accumulation, whereas PPAR gamma agonism targets hepatic inflammation. It has been shown to reduce overall NASH activity score in experimental models of NASH and sonographic improvement in the fatty liver with normalization of liver enzymes in clinical studies.[20,21] In India, Saroglitazar was given approval for the treatment of NASH by the Drug Controller General of India (DCGI) in March 2020. In a trial conducted on 106 patients, Saroglitazar 4 mg was shown to significantly improve alanine transaminase (ALT), liver fat content, insulin resistance and atherogenic dyslipidaemia in patients with NAFLD/NASH; however, improvement in fibrosis was not assessed.[16] This oral drug is now widely used in India for the treatment of NASH.

Although weight reduction and saroglitazar have shown to be effective in NAFLD, there is no Indian study demonstrating the effect of these interventions using noninvasive parameters like liver stiffness measurement (LSM) and controlled attenuation parameter (CAP) using transient elastography. We analysed a cohort of NAFLD patients after intervention with weight reduction advice and saroglitazar for 3 months and evaluated the factors affecting reduction in LSM, CAP score and transaminase levels.

PATIENTS AND METHODS

Study Design and Participants

We prospectively enrolled patients attending the outpatient department of a tertiary care centre in New Delhi from March 2017 to March 2018. Consenting obese (body mass index [BMI] >23 kg/m^2) patients of NAFLD, aged between 18 and 65 years, were included. Fatty liver was diagnosed on ultrasound. Patients with advanced fibrosis/cirrhosis (LSM >13.5 kPa), pregnant patients, major surgery within 3 weeks impairing the ability to do moderate exercise, coronary artery disease, uncontrolled hypertension (systolic BP >160 mm Hg, diastolic BP >100 mm Hg) and uncontrolled diabetes mellitus (HbA1C > 9%) were excluded.

Diagnosis of NAFLD

Participants with fatty liver on ultrasound were further evaluated. Other causes of the fatty liver like HCV hepatitis, disorders of lipid metabolism, severe surgical weight loss (>5% body weight in 3 months before enrolment), drugs causing steatosis (amiodarone, tamoxifen, methotrexate, systemic glucocorticoids, anabolic steroids, tetracycline, oestrogens in doses higher than used in oral contraceptives, vitamin A, L asparaginase, valproate, chloroquine or antiretroviral drugs), Wilson and Celiac disease were ruled out. Subjects then underwent FibroScan® with LSM, median IQR and CAP recorded. Those with an LSM value of more than 13.5 KPa were excluded. Those with a CAP value of more than 248 dB/m were included in the study.

Baseline evaluation and intervention

A detailed history was taken to rule out significant alcohol consumption and secondary causes of fatty liver. Complete hemogram, liver function tests (total and direct bilirubin, aspartate transaminase (AST), ALT, ALP, gamma glutamyl transferase (GGT), serum protein and albumin), renal function tests (blood urea nitrogen, creatinine, calcium, phosphorous, sodium and potassium) and lipid profile (triglycerides, total cholesterol, very low density lipoprotein, low density lipoprotein and high density lipoprotein) were performed in each patient. Relevant investigations like antinuclear antibodies, antismooth muscle antibodies, immunoglubulin G, total iron binding capacity and ferritin were performed to rule out other causes of transaminitis. Basic anthropometry with BMI and waist-to-hip ratio was recorded. A dietician at our centre gave dietary and lifestyle advice, including exercise for weight reduction at the first visit. Saroglitazar 4 mg once a day and other symptomatic treatment was administered. The patients contacted the dietician (telephonically or physically) whenever they required help during the 3-month period. The patient's' follow-up visit with the physician was scheduled as per the need and symptoms of the patients. However, for the purpose of this study, their anthropometry, liver function tests, lipid profile and FibroScan with LSM and CAP value repeated at 3 months. Patients were categorized into those who were able to reduce ≥5% weight and those who could n'ot, and both these groups were compared.

Statistical Analysis

Continuous variables were expressed as median with range and discrete variables were expressed as number (%). Comparison of continuous variables between two groups was done using Mann–Whitney U test and Fisher's exact test or chi-square test was used to compare categorical variables. Wilcoxon signed-rank test was used for paired value comparison. SPSS 23 (Chicago, Illinois) software was used for analysis.

RESULTS

Baseline Patient Characteristics

Of the patients attending outpatient department at a single centre, a total of 112 patients were enrolled for the study after applying inclusion and exclusion criteria. Of these, 91 patients were followed up after 3 months of enrolment and included in the analysis. The median age of the cohort was 45 years (range 18–66 years). There were 74 men (81%) and 17 women (19%). The median BMI was 29.3 kg/

Table 1 Baseline Characteristics of the Patients.

Parameter	Value (*n* = 91)
Age, years	45 (18–66)
Gender, *n* (%)	
Males	74 (81%)
Females	17 (19%)
BMI, kg/m^2	29.3 (23.6–42.2)
AST, IU/dL	40 (22–144)
ALT, IU/dL	48 (13–164)
GGT, IU/dL	42 (4–171)
LSM, kPa	6.7 (3.6–13.1)
CAP, dB/m	308 (249–400)

Note: All values are median (range) or *n* (%).
ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; CAP, controlled attenuation parameter; GGT, gamma-glutamyl transferase; LSM, liver stiffness measurement.

m^2 (23.6–42.2 kg/m^2). The baseline mean AST, ALT, GGT, LSM and CAP values were 40 IU/dL (range 22–144 IU/dL), 48 IU/dL (range 13–164 IU/dL), 42 IU/dL (range 4–171 IU/dL), 6.7 kPa (range 3.6–13.1 kPa) and 308 dB/m (range 249–400 dB/m) (Table 1).

Follow-up

All patients were compliant with the drugs and they tolerated saroglitazar well and there were no major adverse events reported. At 3 months, 57 patients (63%) were able to reduce ≥5% weight, whereas in the remaining 34 patients (37%), the weight reduction was <5% from baseline. The median change in weight in the 57 patients who were able to reduce ≥5% weight was −8 kg (range −15 to −3 kg), whereas the median change in weight in the rest of the 34 patients was just −2 kg (range −2 to +11 kg). The comparison of various parameters in these two groups is shown in Table 2. Transaminases values improved in both the groups; however, LSM and CAP values improved only in patients who reduced weight ≥5%.

DISCUSSION

The main finding of this single-centre, nonrandomized study of saroglitazar in 91 Indian patients of NAFLD presenting to a tertiary care centre, was that saroglitazar was well tolerated and was able to produce improvement in transaminase levels. However, improvement in steatosis and liver stiffness was seen only in patients who were able to reduce at least 5% of their body weight.

NAFLD has emerged as the leading cause of liver disease across the globe and NASH-related cirrhosis is the most common indication of liver transplantation in many parts of the world. However, there are no FDA-approved medications for the treatment of NASH, and weight reduction is the only effective treatment. Saroglitazar has received DCGI clearance for the treatment of noncirrhotic NASH in India.

We advised lifestyle modification and weight loss in all patients. Weight loss through a reduction in caloric intake decreases hepatic free fatty acid supply, improves insulin sensitivity and reduces adipose tissue infliammation.[22] There is improvement in the overall quality of life and histology in NAFLD patients after weight reduction.[23] All patients received 4 mg saroglitazar daily along with weight loss advice. In this study, we found that in overweight patients of NAFLD, a 3-month therapy with saroglitazar is able to reduce transaminases but not LSM and CAP unless accompanied by weight reduction of at least 5% body weight.

Diagnosis of NAFLD is based on a demonstration of intrahepatic fat. While the liver biopsy is the traditional gold standard for the assessment of hepatic necroinflammation and fibrosis, it is not advisable for detecting fat. Transient elastography (FibroScan®, Echosens, Paris) is a noninvasive modality to assess fibrosis by LSM and steatosis by CAP, especially in patients with high BMI, both for the diagnosis and follow-up of NAFLD patients.[24] We used noninvasive modalities of LSM and CAP for assessment of fibrosis and steatosis, respectively. LSM by FibroScan helps to diagnose fibrosis in NAFLD patients.[25] CAP correlates with the degree of steatosis and can also be used to measure the change in steatosis grade on

Table 2 Three-Month Follow-Up of Patients.

	Patients who achieved weight reduction of ≥5% (*n* = 57)			Patients who did not achieve weight reduction of ≥5% (*n* = 34)		
	Baseline	3 months	*P* value	Baseline	3 months	*P* value
Change in weight, kg	–	−8 (−15 to −3)	–	–	−2 (−4 to +11)	–
AST, IU/dL	40 (23–144)	36 (21–88)	**<0.01**	40 (22–110)	35 (21–102)	**0.038**
ALT, IU/dL	53 (17–164)	44 (18–102)	**<0.01**	40 (13–115)	32 (17–102)	**<0.01**
GGT, IU/dL	44 (4–169)	42 (17–86)	0.065	40 (15–171)	36 (11–80)	0.098
LSM, kPa	6.8 (3.6–13.1)	5.9 (3.1–11.9)	**<0.01**	6.3 (3.6–13.1)	6.1 (4.1–14.1)	0.336
CAP, dB/m	311 (251–400)	265 (210–354)	**<0.01**	281 (249–367)	289 (206–396)	0.128

Note: All values are median (range). *P*<0.05 is considered significant.

follow-up. [24,26,27] Karlas *et al* showed CAP value >248 dB/m correlates with the presence of significant steatosis and we used this cut-off to include patients in our study.[28]

Transaminitis is a marker of hepatitis and has some histological correlation with steatosis. We found that while transaminase value reduces, CAP and LSM value does n't reduce significantly after 3 months of saroglitazar treatment unless accompanied by >5% body weight loss. This suggests saroglitazar is ineffective in reducing steatosis or fibrosis in NAFLD after a 3-month treatment. Steatosis is considered as one of the drivers of inflammation in hepatocytes and any treatment of NAFLD should target steatosis reduction. This may signify pathways other than PPAR are responsible for steatosis and need to be targeted. Also, liver stiffness reduction has shown to improve survival in NASH.

Saroglitazar has been shown to improve liver histology in animal models of NASH. Although published clinical data are scarce, there is emerging data from India of its effect in reducing transaminases, LSM and triglyceride levels in dyslipidaemic NAFLD subjects. In an initial case series of 10 patients with diabetic dyslipidaemia and NAFLD, at 9-month follow-up after saroglitazar treatment, significant improvement was observed in shear wave velocity and transaminases levels. Serum TG level was also significantly reduced.[29] Hajare *et al* found significant improvement in transaminitis and liver stiffness after 1 year of saroglitazar treatment in Indian NAFLD patients.[30] The authors of this study did not use CAP values for measuring steatosis and ours is the first study to use CAP values in monitoring steatosis. In another observational study of 30 patients with T2DM and NAFLD treated with saroglitazar for 6 months, significant improvement was observed in glycaemic parameters, liver stiffness and serum transaminase levels.[31] Similarly, Goyal *et al* studied 107 patients with diabetic dyslipidaemia and NAFLD who received saroglitazar 4 mg once daily for 24 weeks. They found that ALT, AST, LSM, CAP, HbA1c and lipid parameters improved significantly. On linear regression, there was a significant association between percent change in ALT and AST with TG reduction after treatment.[32]

Our study confirms the role of significant weight reduction in the treatment of NAFLD and weight reduction and physical activity should remain the cornerstone of any treatment regimen of NAFLD. Saroglitazar controls certain parameters of the metabolic syndrome like cholesterol and triglyceride levels and it may help in decreasing hepatic steatosis or stiffness when associated with weight reduction. The reduction in hepatic steatosis is measured in terms of CAP values in our study. There are no studies comparing CAP values in NAFLD patients after receiving treatment.

There are certain limitations of our study. The study population was predominantly men; however, previous studies did not show any differential effects of saroglitazar based on sex. Also, the diagnosis of NAFLD was based on ultrasound and CAP value, whereas liver biopsy remains the gold standard for the diagnosis of NAFLD. However, biopsy is an invasive procedure with its own risks and complications. CAP has shown to be sensitive and specific for the diagnosis and quantification of hepatic steatosis and thus avoids an invasive procedure. Follow-up of 3 months in our study may be inadequate. This study was an observational study and further randomized studies are required to compare the efficacy of saroglitazar to the established treatment modality like weight reduction.

In conclusion, our single-arm, nonrandomized study showed that in overweight patients with NAFLD, a 3-month therapy with saroglitazar was able to improve transaminases; however, LSM and CAP values improved only when patients were able to reduce their weight by at least 5%. Furthermore, larger randomized controlled trials are needed to document the independent effect of saroglitazar in these patients.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

AA conceptualized and supervised the study; PP recruited the patients, did data collection, and wrote the manuscript; AK did statistical analysis; PS, PC and NV gave intellectual inputs and revised the manuscript.

CONFLICTS OF INTEREST

The authors have none to declare.

ACKNOWLEDGEMENT

This study did not receive any funding.

FUNDING

None.

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