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ORIGINAL RESEARCH ARTICLE

The role of saroglitazar in the treatment of metabolic dysfunction-associated steatohepatitis in non-diabetic patients: A prospective observational study

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Abstract

Metabolic dysfunction-associated steatohepatitis (MASH) is a metabolic disease characterized by hepatic fat accumulation and significant inflammation. Patients with hepatic steatosis who also have at least one of the five cardiometabolic risk markers are considered to have metabolic dysfunction-associated steatotic liver disease. MASH is diagnosed when a microscopic examination of liver tissue reveals fat accumulation in hepatocytes along with inflammation and destruction of liver cells. This study aims to assess the role of saroglitazar, a dual proliferator peroxisome-activated receptor agonist, in the medical therapy of MASH in non-diabetic patients. A prospective observational study was carried out in a tertiary care facility in north India. A total of 51 non-diabetic MASH patients (males and females, 18 – 75 years of age, body mass index ≥18.5 kg/m^2) were included in this study. Diagnosis of MASH was based on liver FibroScan (controlled attenuation parameter [CAP] score >238, liver stiffness measurement [LSM] value >7kPa) along with raised liver enzymes (serum glutamic-oxaloacetic transaminase [SGOT], serum glutamic-pyruvic transaminase [SGPT] > upper value of normal limits). All these 51 patients received 4 mg once-daily dose of saroglitazar for the treatment of MASH for 24 weeks. In this study, a standard treatment protocol was used. The percentage changes in aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase, triglyceride, low-density lipoprotein (LDL), and LSM with CAP were evaluated using an analysis of variance test and multiple regression analysis. About 72.5% of these patients were male and 27.5% were female (male: female ratio of 2.6). The mean age of patients was 51 ± 13.13 years. Pre- and post-treatment values of different parameters were compared. Pre-treatment mean values of ALT and AST were 93.83 ± 6.16 U/L and 76.13 ± 4.1 U/L, respectively, while their post-treatment mean values were 32.97 ± 2.15 U/L and 34.57 ± 1.65 U/L, respectively (*P*-value for AST and ALT <0.001). In conclusion, saroglitazar is effective in the medical management of MASH by reducing liver stiffness and suppressing elevated liver enzymes (SGOT/SGPT) as well as serum LDL levels over 6 months.

Keywords: Hepatic steatosis; Hepatocytes; Metabolic dysfunction-associated steatohepatitis; Proliferator peroxisome activated receptor; Saroglitazar

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1. Introduction

Metabolic dysfunction-associated steatohepatitis (MASH) is a metabolic disease marked by hepatic fat buildup and significant inflammation. From metabolic dysfunction-associated fatty liver to MASH, metabolic dysfunction-associated steatotic liver disease (MASLD) encompasses a wide range of chronic liver diseases.[1] Significant data has surfaced in the last 20 years demonstrating the worldwide occurrence of MASLD and linking it to a range of liver conditions, including those that can lead to cirrhosis and hepatocellular carcinoma. Most of the cases with MAFLD are asymptomatic and are diagnosed incidentally.[2]

A severe type of MASLD that affects 35 million individuals worldwide, MASH is linked to hepatocellular inflammation as a result of fat buildup in the cells. MASLD significantly adds to the worldwide burden of illness. The prevalence of MASLD is 25.24% worldwide, with South America and the Middle East having the highest rates. In India, the prevalence of adult MASLD has been reported between 6.7% and 55.1%, whereas that of pediatric MASLD has been reported between 7.3% and 22.4%. Obesity, type 2 diabetes, dyslipidemia, hypertension, and metabolic syndrome are the common metabolic comorbidities linked to MASLD. It has been estimated that 33.5% of adults worldwide will be affected by MASLD and another 27% will have MASH by 2030.

MASLD-related hepatocyte steatosis is typified by the accumulation of fat droplets in the hepatocytes. MASH may occur from hepatocellular damage and inflammation, either in conjunction with or apart from hepatic fibrosis. Factors including insulin resistance, oxidative stress, inflammation, altered adipokine and cytokine secretion, altered lipid metabolism, and endoplasmic reticulum stress are associated with these disorders. These characteristics make MASH a clinically aggressive form of MASLD, and individuals with MASH are more likely to develop cirrhosis. As a result, for the past several years, attempts have been made to study and develop drugs specifically for this condition.

The primary diagnostic subtype of MASLD is MASH, which carries a risk of cirrhosis and liver-related consequences. However, it is important to note that all subtypes of MASLD elevate the likelihood of cardiovascular events and mortality. Projections suggest a steep increase in the prevalence of MASLD patients in the coming years, driven by the growing rates of obesity and type 2 diabetes.[3,4] MASLD often emerges in individuals with a medical history of diabetes and obesity. Our understanding of MASLD's etiology and progression has significantly expanded in recent decades. The well-known "multiple hit hypothesis" suggests that insulin resistance is essential for the development of multiple sclerosis lipogenesis (MASLD) by increasing the liver's ability to use free fatty acids, which in turn causes fatty liver and lipotoxicity.[5,6]

The American Association for the Study of Liver Diseases recommendations emphasize that only biopsy-confirmed MASH warrants medical intervention[7,8] While various medications have been explored for MASH treatment, none have received official approval.[7] Among the medications considered are glitazones, with pioglitazone being one example. Studies have shown that pioglitazone can improve NAFLD activity score, lead to MASH resolution, and enhance fibrosis, along with improving histological aspects like steatosis, inflammation, and hepatocellular ballooning.[9,10]

In MASLD, the primary goal of pharmacological intervention is to manage liver inflammation and fibrosis. This is due to the fact that in individuals with MASLD, the degree of liver inflammation and fibrosis is strongly linked to both extrahepatic and hepatic morbidity and mortality. A dual proliferator peroxisome activated receptor (PPAR)-α/γ agonist called saroglitazar magnesium was created to lessen the negative effects of selective PPAR-γ agonism. For saroglitazar, the proposed mechanism of action involves targeting PPAR-α and PPAR-γ, which leads to an enhancement in insulin sensitivity and lipid oxidation. This dual mechanism reduces lipotoxicity by decreasing fat accumulation in the liver and improving fat metabolism there.[11,12] It inhibits the production and release of triglycerides (TG), increases the hepatic oxidation of fatty acids, and through PPAR-α agonism, improves the levels of lipoproteins in circulation. Furthermore, through PPAR-γ agonism, saroglitazar decreases blood glucose and glycosylated hemoglobin (HbA1c) levels, enhances insulin sensitivity, and controls the transcription of genes that respond to insulin. In India, saroglitazar has been licensed for the treatment of hypertriglyceridemia and diabetic dyslipidemia since 2013. In addition, it has been authorized in some other countries, to treat hypertriglyceridemia and dyslipidemia in type 2 diabetic patients whose condition is not managed by statins.

Numerous investigations, including human and animal MASLD models, have demonstrated its effectiveness in reducing hepatic steatosis, hepatocellular inflammation, and fibrogenic activity.[13,14] Saroglitazar has been shown in animal models to decrease alanine aminotransaminase (ALT) levels and to alleviate hepatic steatosis, hepatocellular ballooning, and lobular inflammation in MASH caused by a high-fat, choline-deficient diet or Western diet. Moreover, saroglitazar was shown to significantly lower insulin resistance, TG, total cholesterol, and metabolically

active lipid species, including diglycerides, ceramides, and sphingomyelins, as well as improve liver fibrosis and inflammation biomarkers in mice with Western diet-induced MASH. Saroglitazar has been shown in clinical trials to be useful for people with MASLD; however, there are very few prospective studies dedicated to studying the role of saroglitazar in the management of MASLD/MASH.

2. Materials and methods

The current prospective observational study was conducted at the Max Hospital in Vaishali, Ghaziabad, India, a tertiary care facility in northern India, for a duration of 6 months, from November 2024 to May 2023. The Indian Council of Medical Research's Good Clinical Practice and Declaration of Helsinki guidelines were adhered to in this investigation. A total of 51 non-diabetic MASH patients (males and females, 18–75 years of age) were prospectively followed up in this study. Diagnosis of MASH was done based on liver FibroScan (controlled attenuation parameter [CAP] score >238, liver stiffness measurement [LSM] value >7kPa) along with raised liver enzymes (serum glutamic-oxaloacetic transaminase [SGOT], serum glutamic-pyruvic transaminase [SGPT] > upper value of normal limits). All of these 51 patients received 4 mg once-daily dose of saroglitazar for the treatment of MASH from the hepatology and gastroenterology clinic of the hospital. In the current study, standard treatment protocols were used. Analysis of variance (ANOVA) test and multiple regression analysis were used to assess the percentage change in body mass index (BMI), aspartate transaminase (AST), ALT, bilirubin, TG, low-density lipoprotein (LDL), and LSM with CAP.

2.1. Sample size calculation

Fifty patients were proposed for this study. The primary outcome of interest is the change in ALT level from before therapy to after therapy. This parameter gives a standard deviation (SD) of 15.24 (δ = 15.24), which allows the detection of a change of at least δ = 5 unit/L. This indicates that the minimum sample size of 50 is required to ensure the reliable detection of ALT level changes from before therapy to after therapy.

2.2. Inclusion criteria

The participant inclusion criteria for this study are as follows:

(i) Males or females
(ii) 18–75 years of age
(iii) BMI ≥18.5 kg/m^2
(iv) Non-diabetics (fasting blood sugar <100 mg/dL, HbA1C <5.7 and post-prandial blood sugar <140 mg/dL)
(v) Confirmation of hepatic steatosis through imaging or histologic means
(vi) No history of significant alcohol consumption
(vii) No competing etiologies for hepatic steatosis
(viii) No co-existing causes for chronic liver disease
(ix) Having elevated AST/ALT levels along with liver stiffness value ≥7 kPa and/or liver steatosis CAP >238 dB/m, measured through liver FibroScan.

Patients who received saroglitazar treatment for 24 weeks and came for follow-up during and at the end of therapy were analyzed.

2.3. Exclusion criteria

Exclusions from our study included patients with a history of type 2 diabetes or who had taken diabetic medications within the 6 months before recruitment, dyslipidemia history, the presence of chronic hepatitis B or C infection (positive HbsAg or anti-HCV-ab), evidence of chronic liver disease on abdominal ultrasound and portal Doppler, significant alcohol intake (>20 mg/day for males and >10 mg/day for females), and a history of anti-obesity medication intake in the 6 months before recruitment. In addition, cases who were having a history of drug use that resulted in hepatic steatosis/fibrosis including psychotropic drugs, or cases with a history of hepatotoxic drug consumption were also excluded. Individuals with additional comorbid conditions such as hypothyroidism, ischemic illness, or chronic kidney disease were excluded. The study also excluded the patients with any confounding factors that could cause the FibroScan values to be overestimated, such as liver congestion, ascites, liver inflammation from alcohol consumption or recent liver illness, benign or malignant liver tumors, biliary obstructions, *etc.* Those with a history of severe illness or other conditions that would make the patient, in the investigator's opinion, unsuitable for the study, such as those who have a known allergy, sensitivity, or intolerance to saroglitazar or formulation ingredients, women who are pregnant, nursing, or who may become pregnant in the future but are not using appropriate contraceptive measures, were also excluded from the study.

2.4. Data collection method

All patients underwent general physical examination including recording of vitals. The patients were enrolled and selected as per inclusion and exclusion criteria. If the patient was suffering from any other concomitant diseases while receiving treatment for the same, the details were recorded in a case reporting form. The primary efficacy variable was the mean change in NAFLD fibrosis score at the end of the study (week 24) as compared with baseline; the mean change in TG, LDL, high-density lipoprotein, and cholesterol levels; and the mean change in liver function test (LFT) levels and BMI

values. Every participant was interviewed using a series of questionnaires covering demographic characteristics such as age, gender, anthropometric measurement, family history of metabolic disorders or liver disease, current medications, comprehensive medical history including history of alcohol consumption, and any comorbidities such as diabetes, hypertension, and dyslipidemia. The whole physical examination was performed by a skilled technical assistant. A regular stadiometer was used to measure height, and a typical bathroom scale was used to assess weight. The BMI was computed using weight in kilograms and height in meters. Baseline demographics of patients were documented, including age, gender, height, weight, waist circumference, comorbid conditions, and current medicines.

Comprehensive medical history was documented, encompassing the individual's history of alcohol consumption, concurrent medication use, and metabolic comorbidities such as obesity, hypertension, diabetes mellitus, coronary artery disease, and cerebrovascular disease.

2.5. Statistical analysis

ANOVA test was performed to compare the data collected before and after treatment, and multiple regression analysis was used to determine the percent change in ALT, AST, alkaline phosphatase (ALP), bilirubin, BMI, LSM, and CAP. The collected data were entered into Microsoft Excel 2010 for statistical analysis, which was carried out with the aid of Statistical Package for the Social Sciences software. Categorical data are presented as proportions, and continuous data are expressed as mean ± SD (for parametric data) or median and interquartile range (for non-parametric data).

3. Results

A total of 72 individuals were screened for the study. After excluding 21 patients who met one or more of the outlined exclusion criteria, only 51 patients were included in the protocol analysis. Baseline investigations of all 51 patients were conducted. Table 1 displays the basic demographic characteristics of the patients.

All 51 patients who were treated with saroglitazar at 4 mg once-daily dose for 24 weeks were followed up till the end of the study. Out of the sample, 34 patients (72.5%) were male and 17 (27.5%) were female (male-to-female ratio of 2.6). The mean age of patients was 51 ± 13.13 years. The mean value of the fasting blood sugar level was 92.6 ± 9.7 mg/dL. On baseline investigation, the mean value of BMI was 24.1 ± 3.9. LFT and FibroScan procedures (two variables, *i.e.*, LSM and CAP, were noted) were done for the diagnosis of MASH. At the baseline visit, there was an increased level of liver enzymes such as ALT, AST, and gamma-glutamyl transferase (GGT). Comparisons of pre- and post-treatment values of different laboratory parameters including liver enzymes such as ALT, AST, ALP, and GGT, as well as lipid profile and FibroScan parameters such as LSM and CAP, were done. After treatment with saroglitazar at 4 mg once-daily dose for 24 weeks, we detected a significant decrease in values of ALT (32.97 ± 2.15 U/L; $P < 0.001$), AST (34.57 ± 1.65 U/L; $P < 0.001$), GGT (31.86 ± 2.37 U/L). Mean values of pre-treatment ALT and AST were 93.83 ± 6.16 U/L and 76.1 ± 4.1 U/L, respectively, while mean values of post-treatment ALT and AST were 32.97 ± 2.15 U/L and 34.57 ± 1.65 U/L, respectively (both $P < 0.001$). The mean values of pre-treatment FibroScan parameters such as LSM and CAP were 16.2 ± 1.57 kPa (kilopascal) and 297.80 ± 37.38 dB/m (decibel/meter), whereas post-treatment mean values were 5.67 ± 0.23 kPa and 264.80 ± 6.81 dB/m, respectively (LSM and CAP: $P < 0.001$ and $P = 0.049$, respectively). The average values of pre- and post-treatment LDL were 122.1 ± 4.84 and 93.51 ± 4.12, respectively ($P < 0.001$). A significant decrease in values of LSM and CAP ($P < 0.001$ and $P = 0.049$, respectively) indirectly showed that there was a marked improvement in fibrosis score. The comparison of BMI values between the pre- and post-treatment groups showed no significant change (pre- and post-treatment BMI values were 24.1 ± 3.9 and 24.5 ± 3.05, respectively; $P = 0.565$).

It was noticed that saroglitazar, at a once-daily dose of 4 mg, was safe and well-tolerated because there were no serious adverse events associated with the medication that required treatment cessation. All of the 51 patients completed the follow-up for 6 months and were included in the post-treatment data analysis.

4. Discussion

The purpose of this trial was to evaluate saroglitazar's effectiveness and tolerability in non-diabetic patients with hepatic inflammation and fibrosis as detected on liver FibroScan/elastography. This study found that saroglitazar is safe, tolerable, and effective in the management of MASH. Information about saroglitazar's effectiveness in the Indian population is limited. In this study, liver enzyme and FibroScan parameter improvements before and after therapy were compared in non-diabetic individuals with MASH to determine the efficacy of saroglitazar. Based on the widely accepted recommendations that have been backed by available evidence, all patients were given 4 mg of saroglitazar.[15] Since this is the first study of its kind in India to highlight the potential advantages of saroglitazar, particularly in non-diabetic MASH, it adds considerably to the current medical knowledge.[16]

Table 1. Comparison of demographic, biochemical, and FibroScan values

Parameters	Pre-treatment (mean±SD)	Post-treatment (mean±SD)	*P*-value
Age (years)	51±13.13		
Gender (male: Female)	34:17		
Fasting blood sugar (mg/dL)	92.6±9.7		
BMI	24.1±3.9	24.5±3.05	0.565
ALT (U/L)	93.82±6.16	32.97±2.15	<0.001
AST (U/L)	76.13±4.1	34.57±1.65	<0.001
ALP (U/L)	92.87±4.34	89.59±2.48	0.262
Total bilirubin (mg/dL)	0.90±0.06	0.79±0.05	0.032
Serum protein (mg/dL)	7.2±0.08	7.23±0.08	0.932
Serum albumin (g/dL)	4.5±0.05	4.47±0.05	0.776
LDL (mg/dL)	122.1±4.84	93.51±4.12	<0.001
HDL (mg/dL)	44.47±1.14	46.70±1.25	0.211
Non-HDL (mg/dL)	162.15±4.5	162.15±4.5	0.999
Triglyceride (mg/dL)	232.02±42.38	157.48±2.98	0.086
LSM (kPa)	16.2±1.57	5.67±0.23	<0.001
CAP (dB/m)	297.09±37.38	264.80±6.81	0.049
GGT (U/L)	74.77±5.86	31.86±2.37	<0.001

Note: $P<0.05$ is considered statistically significant.
Abbreviations: ALP: Alkaline phosphatase; ALT: Alanine transaminase; AST: Aspartate transaminase; BMI: Body mass index; CAP: Controlled attenuation parameter; GGT: Gamma-glutamyl transferase; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; LSM: Liver stiffness measurement; SD: Standard deviation.

Many studies emphasize the significance of pinpointing the optimal pharmaceutical target for the management of mixed hepatic liver disease (MASLD) by addressing multiple aspects of the condition, such as enhancing insulin sensitivity, decreasing hepatocellular inflammation, lowering oxidative stress, addressing mitochondrial dysfunction, and addressing liver fibrosis.[17] With little success, only a number of medications have previously been demonstrated to exhibit efficacy in the treatment of MASLD. Peroxisome proliferator-activated receptors (PPARs) are essential for maintaining lipid homeostasis. PPARα is primarily expressed in hepatocytes and inhibits the accumulation of fatty acids within the liver, preventing steatosis and steatohepatitis. On the other hand, PPARγ is primarily found in adipocytes and improves insulin sensitivity while lowering the availability of fatty acids to the liver.[18]

Medications with antifibrotic qualities, such as pioglitazone (a PPARγ agonist), are reportedly known to improve histology outcomes in MASLD patients. However, because of a number of adverse effects, including weight gain, heart failure, headaches, impaired vision, and bladder cancer, they were not authorized for use in the treatment of MASLD.[19] Furthermore, fibrate PPARα agonists did not show encouraging outcomes in MASLD studies.[12] The efficacy of a PPAR-α/δ agonist called elafibrinor is presently being tested in individuals with MASLD.[20] While metformin, ursodeoxycholic acid, and Vitamin E were once utilized to treat MASLD, more recent research has shown that these medications had no therapeutic benefits.[7,8] Although more extensive research is required to evaluate the safety and effectiveness of obeticholic acid, encouraging information has surfaced regarding its involvement in MASLD patients.[21]

Recently, saroglitazar – a dual PPARα/γ agonist – was licensed to treat MASLD. It has demonstrated potential in lowering alterations in dyslipidemia, decreasing glucolipotoxicity to reduce insulin resistance, and having an agonistic impact on PPARγ, particularly in those with diabetes and dyslipidemia.[20] Notably, in the absence of a histological investigation, increased levels of ALT and AST – markers of hepatic inflammation – play a critical role in the diagnosis of MASH.

The degree of liver damage has historically been determined by quantifying the levels of liver enzymes. Due to its non-specificity and lack of correlation with fibrosis, this method has intrinsic limitations. However, due to the low cost involved and wide availability, it is still broadly utilized, offering substantial assistance in real-world situations. In this study, ALT and AST levels

were dramatically lowered after 24 weeks of therapy, suggesting that saroglitazar is useful in reducing liver inflammation, substantiating its efficacy based on a non-alcoholic steatohepatitis population.[12] These findings are consistent with previous research, such as that conducted by Kaul *et al.*[21] which reported a noteworthy decrease in ALT following 12 – 58 weeks of saroglitazar treatment, and another[15] which demonstrated a reduction of 60% in ALT and 43% in AST in animal models with MASLD after 12 weeks of saroglitazar treatment. Similarly, Goyal *et al.* observed significant reductions in ALT and AST after using saroglitazar for 24 weeks, concluding that saroglitazar can improve hepatic inflammation and resolve transaminitis.[20]

The ultimate goal of pharmacological treatment is to reverse liver fibrosis. However, a liver biopsy is usually required for histopathological examination to accurately detect fibrosis, and this procedure is invasive and impractical.[12] Liver stiffness measurement, which leverages FibroScan as a substitute non-invasive approach for evaluating liver fibrosis and cirrhosis, has demonstrated potential in a number of recent investigations.[22,23] According to LSM measurements utilizing FibroScan, the current study found a substantial decrease in fibrosis. Although in our study we did not compare the fibrosis score directly between pre- and post-treatment groups, a significant decrease in LSM (kPa) value indirectly indicates improvements in liver fibrosis and cirrhosis. Goyal *et al.* also noted a substantial decrease in LSM after 24 weeks of saroglitazar treatment,[20] and another study reported a reduction in liver fibrosis, measured by shear wave elastography, following 9 months of saroglitazar treatment.[24]

In addition to fibrosis assessment, evaluating changes in liver fat content is an essential parameter for gauging the response to MASLD therapy. Although abdominal ultrasonography can be used as a screening method to diagnose fatty liver, it is not very effective in identifying changes in the modest amount of liver fat. With high accuracy in detecting changes in liver fat content, the CAP, as evaluated by FibroScan, provides an acceptable option for diagnosing fatty liver.[12] The analysis of the current study showed a considerable decrease in liver fat, as determined by CAP utilizing FibroScan. According to Goyal *et al.*[20] and Kaul *et al.*,[21] there was a noteworthy improvement in liver fat, as evaluated by CAP, following 12 – 58 weeks of saroglitazar therapy in MASLD, which is consistent with our findings. It has also been reported that CAP values significantly decreased after 24 weeks of saroglitazar treatment.[20]

Very few studies have been conducted to validate the role of saroglitazar in MASH management in non-diabetic patients. It should be highlighted that the sample used, the prospective nature of this study, and the use of non-invasive methods for assessing hepatocellular inflammation and fibrosis are the strengths of this study, despite some limitations. The small sample size and the brief follow-up period were the main limitations of this study. It is a larger sample study covering an extended period of follow-up that would be conducted in the future to provide more in-depth insights. The inability of this study to forecast advantage over other compounds now under investigation for MASH is also a limitation. In addition, there was a lack of follow-up information about whether stopping the medication would result in a reversal of the biochemical and stiffness parameter improvements or a continued benefit. However, the study did not address this crucial question owing to the research design employed. The assessment of cardiovascular outcome was not conducted as well. There is a strong association between MASH and cardiovascular incidents, and the majority of causes of death and morbidity in the MASH population are cardiovascular in nature; however, the present study did not evaluate any cardiovascular parameters, neither in the pre- nor post-treatment context. Hence, it is highly anticipated that a long-term follow-up study will be conducted in the future to determine whether there is an increase or decrease in cardiovascular outcomes, a research topic this study was not intended to cover. Although underweight patients were excluded from this study, the relationship of sarcopenia with outcomes in this study was not explored, making it one of the limitations of this study.

It is important to note that although the adverse effects of saroglitazar such as hypoglycemia, nausea, and chest pain have been mentioned in the literature, none of these side effects were reported by the patients taking part in this study. Similar research using animal models did not find any adverse consequences.[25] Moreover, no major negative effects were reported in human studies.[26] The results of this study support the idea that individuals receiving medication for MASLD may tolerate saroglitazar well.

5. Conclusion

Saroglitazar has a beneficial impact on the medical management of MASH by reducing liver inflammation through normalization of liver enzymes and reduction of liver stiffness. It also curtails fat buildup in hepatocytes, which is manifested in terms of the significant decrease in CAP value. Saroglitazar also lowers the elevated levels of liver enzymes (SGOT/SGPT) as well as serum LDL levels within a duration of 6 months. The findings of this study ascertain saroglitazar as a viable treatment option for MASH management. Nonetheless, further research involving a larger sample and longer follow-up period,

with an emphasis on sarcopenia and advanced cirrhosis, is warranted to help validate saroglitazar as a primary drug for this liver disease.

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Conflict of interest

The authors declare they have no competing interests.

Author contributions

Conceptualization: Premashish Kar, Priyanshu Bhardwaj, Jata Shankar Kumar
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Ethics approval and consent to participate

The study was approved by the Institutional Ethics Committee, Max Super Specialty Hospital in May 2022. Written informed consent was taken from all the patient before their participation.

Consent for publication

All participants in the study gave their informed written consent for their data to be published.

Availability of data

Data are available from the corresponding author upon reasonable request.

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