

ORIGINAL RESEARCH ARTICLE



Comparative Effects of Saroglitazar, Vitamin E and Lifestyle Modification on Liver Fibrosis Markers in Metabolic Dysfunction-Associated Steatotic Liver Disease

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Abstract

Introduction: This study presents the effects of metabolic dysfunction-associated steatotic liver disease (MASLD), which is a global health issue. It is related with obesity, metabolic dysfunction, and progressive liver fibrosis. In order to prevent this issue, it requires an effective treatment plan. This study assessed the effect of Saroglitazar, Vitamin E, and lifestyle modification on liver fibrosis markers and metabolic parameters in patients with MASLD/metabolic dysfunction-associated steatohepatitis (MASH).

Methods: This investigator-initiated, prospective, randomised study was conducted among 170 patients from May 2025 to April 2026 in our hospital. 170 participants were divided into three groups: Saroglitazar (n=60), Vitamin E (n=60), and Only Lifestyle group (n=50). Each parameter was measured at baseline and after 24 weeks.

Results: Baseline characteristics were comparable across the Saroglitazar (n=60), Vitamin E (n=60), and Lifestyle (n=50) groups. Median age (33–35 years; $p=0.72$), male distribution (86–88%; $p=0.91$), anthropometric measures, and all baseline laboratory parameters showed no significant differences (all $p>0.05$). After 24 weeks, improvements were observed across all groups, with significant between-group differences only for triglyceride reduction, which was greatest with Saroglitazar (-14.2 vs. -4.5 vs. -13.5 mg/dL; $p=0.034$). The proportion achieving ≥ 2 kPa liver stiffness reduction was highest with Saroglitazar (40%), followed by Vitamin E (35%) and Lifestyle intervention (20%), with Saroglitazar significantly outperforming Lifestyle intervention ($p=0.028$).

Conclusion: The study concluded that the Saroglitazar showed improvement in the triglyceride levels and liver stiffness response, while all other three interventions showed similar improvement in the fibrosis and the functional marker for liver.

Key words: Metabolic dysfunction-associated steatotic liver disease, MASLD, Saroglitazar, Vitamin E, Liver fibrosis

1 | INTRODUCTION

Metabolically dysfunction-associated steatotic liver disease (MASLD) is defined as steatosis involving 5% of hepatocytes, with at least one of the five cardio-metabolic risk factors after exclusion of secondary causes of hepatic steatosis (1). The prevalence of MASLD is 32% among adults globally, with a higher prevalence

among males (about 40%) than females (about 26%) (2). The prevalence rate of MASLD in India is estimated to range from 9% to 53% across several studies and is related to metabolic syndrome (3). MASLD includes simple steatosis, metabolic dysfunction related to steato-hepatitis, cirrhosis, and hepatocellular carcinoma. The interventions in MASLD must be provided prior to the development of the cirrhosis and its complications. The manage-

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ment of MASLD involves lifestyle intervention, and a range of different pharmacological agents targeting the metabolic impairments, inflammation, and associated fibrosis. Lifestyle interventions are a significant component for managing MASLD. Lifestyle management of MASLD involves the lifestyle interventions which include changes in exercise and dietary patterns. Various dietary approaches have been evaluated, including hypocaloric diet, the mediterranean diet and the intermittent fasting (4). Resmetirom is a thyroid hormone receptor- β agonist and has been approved by the US Food and Drug Administration (USFDA, March 2024) for the treatment of the MASLD/MASH (5). Pioglitazone is an insulin sensitizer, which has been widely used for the treatment of type 2 diabetes mellitus, which is related to the MASLD/MASH, due to its ability to improve insulin sensitivity, reduce hepatic steatosis and improve histological outcomes. (6). Vitamin E has been used for its antioxidant properties and to reduce liver inflammation. This also improved the liver histology in biopsy among non-diabetic MASH patients (7). Saroglitazar is a dual peroxisome proliferator that activates the receptor α/γ agonist and has been utilised for the treatment of dyslipidaemia and reduces the hepatic fat content as well as inflammation (8). Due to the absence of the therapeutic agent for the management of MASLD, Saroglitazar, along with the lifestyle intervention, is endorsed by the Indian National Association for the study of the Liver (INASL) for the management of the MASLD (9, 10). Another study noted that Vibration-controlled transient elastography (TE) (Fibro Scan), which is an invasive technique for the assessment of the hepatic fibrosis and steatosis, has been observed as the most accurate and reliable non-invasive method for the assessment of liver fibrosis and steatosis among patients with NAFLD (11). The prevalence of MASLD in Indian population is estimated between 9-53% in different studies and often associated with metabolic syndrome (12). The aim of the study is to compare the impact of Saroglitazar, Vitamin E, and modification in the lifestyle on the liver fibrosis markers in metabolic dysfunction-associated Steatotic Liver Disease.

2 | METHOD

Research Design

This is an investigator-initiated, prospective, randomised study that was conducted in our hospital in India. This study was conducted among 170 patients from May 2025 to April 2026. The significance of this study, it was to compare the effectiveness of Saroglitazar, Vitamin E, and Lifestyle Modification on liver fibrosis markers and metabolic dysfunction, which is associated with Steatotic Liver Disease. This study considered participants aged from 19 to 55 years who were diagnosed with metabolic dysfunction-associated steatotic liver disease (MASLD) or metabolic dysfunction-associated steatohepatitis (MASH). In addition, this study excluded those participants who had diabetes mellitus, alcohol intake, hepatitis B or C infection, autoimmune or other chronic liver diseases, decompensated cirrhosis, thyroid disorders, chronic kidney disease, or any serious systemic illness. In addition, this study also excluded participants such as pregnant and lactating women and patients taking alternative medicines or other drugs that could affect liver function. Lifestyle interventions consisted of dietary advice to reduce the intake of high-carbohydrate and high-glycaemic-index foods, along with regular physical activity. All participants were encouraged to follow a healthy diet and perform brisk walking for 30–40 minutes daily throughout the study period. Participants were regularly followed up during the study period, and adherence to lifestyle recommendations was reinforced during follow-up visits or through telephone contact. Baseline demographic, clinical, and laboratory parameters were recorded before the intervention. Furthermore, relevant clinical and laboratory assessments were repeated to assess treatment outcomes. Participants were also observed for compliance with lifestyle changes and for the occurrence of any adverse events during the follow-up period.

Inclusion Criteria

- This study included patients aged from 19 to 55 years.
- This study included those diagnosed with metabolic dysfunction-associated steatotic liver disease (MASLD) or metabolic dysfunction-associated steatohepatitis (MASH).

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• This study included participants who were willing to participate in the study.

Exclusion Criteria

- Patients with diabetes mellitus.
- History of significant alcohol consumption.
- Presence of hepatitis B or hepatitis C infection.
- Patients with autoimmune liver disease or other chronic liver disorders.
- Patients with decompensated cirrhosis.
- Patients with thyroid disorders.
- Patients with chronic kidney disease.

Statistical Analysis

The sample size was calculated by using a statistical tool which helped to estimate comparisons between study groups. Statistical analysis was performed using IBM SPSS Statistics version 27.0. Allocation concealment was maintained through sealed, opaque envelopes containing the group assignments. In this study, treatment allocation was randomized, and eli-

gible participants were assigned to the respective study groups.

3 | RESULTS

Presents the demographic, clinical, and laboratory details of participants who were participants in this study. The median age of patients ranged from 33 to 35 years, with no significant variance among the groups ($p=0.72$). The percentage of male participants was equal among these 3 groups (86–88%), and the p -value was 0.91. In addition, these parameters measure height, weight, and body mass index (BMI), and did not differ significantly between the study arms (all $p>0.05$). Laboratory parameters, such as haemoglobin, total leucocyte count, platelet count, liver enzymes (AST and ALT), total bilirubin, total protein, serum albumin, alkaline phosphatase, and triglyceride levels, were also comparable among the three groups at baseline (all $p>0.05$).

Table 1. Baseline Characteristics of All Patients (Total N = 170)

Parameters	Saroglitazar group (N=60)	Vitamin E group (N=60)	Only Lifestyle group (N=50)	P value
Age (yr)	33.00 ± 9.63	35.00 ± 10.37	34.00 ± 8.15	0.72
Gender, Male, n (%)	53 (88.33%)	88 (146.66%)	86 (172%)	0.91
Height (m)	1.68 ± 0.10	1.68 ± 0.08	1.66 ± 0.09	0.28
Weight (kg)	80.20 ± 12.44	75.50 ± 11.63	75.30 ± 13.07	0.18
BMI (kg/m ²)	27.72 ± 3.28	26.90 ± 3.37	27.40 ± 3.71	0.58
Haemoglobin (g/dL)	13.86 ± 1.41	13.78 ± 1.49	14.05 ± 1.53	0.62
Total leucocyte count (thousands/ μ L)	8080 ± 1865	7320 ± 2180	7720 ± 2045	0.09
Platelet count (thousands/ μ L)	188.00 ± 43.70	180.00 ± 44.81	171.00 ± 35.56	0.06
Serum AST (IU/L)	54.10 ± 21.11	52.40 ± 22.52	55.80 ± 30.07	0.87
Serum ALT (IU/L)	89.10 ± 42.37	84.95 ± 32.44	75.10 ± 43.78	0.41
Total bilirubin (mg/dL)	0.72 ± 0.36	0.70 ± 0.30	0.70 ± 0.24	0.64
Total protein (g/dL)	7.96 ± 0.60	7.82 ± 0.39	7.94 ± 0.61	0.34
Serum albumin (g/dL)	4.62 ± 0.40	4.55 ± 0.30	4.60 ± 0.42	0.63
Serum alkaline phosphatase (IU/L)	136.90 ± 64.89	132.60 ± 53.41	144.30 ± 151.04	0.29
Serum triglycerides (mg/dL)	177.90 ± 59.85	179.10 ± 65.19	186.10 ± 107.33	0.55
Liver stiffness measurement (kPa)	6.80 ± 2.07	7.10 ± 2.59	6.70 ± 2.07	0.42
Controlled attenuation parameter	300.80 ± 43.11	297.50 ± 35.93	303.80 ± 50.81	0.81
FIB-4	1.02 ± 0.50	1.19 ± 0.54	1.29 ± 0.74	0.07
APRI	0.70 ± 0.36	0.77 ± 0.38	0.78 ± 0.42	0.49
NAFLD fibrosis score	-2.82 ± 0.70	-2.63 ± 0.92	-2.45 ± 1.01	0.11

* Parametric data (mean ± SD)# Non-parametric data

Displays the variations among the Saroglitazar, Vitamin E, and Lifestyle Intervention groups after 24 weeks of treatment. The most of the result variables displayed improvements in all three groups; however, there were no statistically significant differences between the groups for NAFLD fibrosis score ($p=0.39$), liver stiffness measurement ($p=0.21$), weight ($p=0.15$), BMI ($p=0.22$), serum AST ($p=0.46$), serum ALT ($p=0.30$), controlled attenuation parameter ($p=0.89$), FIB-4 score ($p=0.47$), and APRI ($p=0.95$). Although the Saroglitazar group presented numerically greater reductions in weight, BMI, AST, and ALT levels, these

differences were not statistically significant compared with the other groups. A significant difference was observed only for serum triglyceride levels ($p=0.034$). The Saroglitazar group demonstrated the extreme level of reduction in triglycerides [-14.2 mg/dL (IQR: -60.0 to -18.5)], whereas the Lifestyle Intervention group [-13.5 mg/dL (IQR: -126.0 to 3.0)], and the Vitamin E group exhibited a comparatively smaller reduction [-4.5 mg/dL (IQR: -29.0 to 48.5)]. Moreover, this study presents that Saroglitazar may have a more positive effect on triglyceride reduction instead of Vitamin E or lifestyle intervention alone.

Table 2. Comparison of Outcome Changes After 24 Weeks

Variables	Saroglitazar group N=60	Vitamin E group N=60	Only Lifestyle group N=50	P value
NAFLD fibrosis score*	0.06 ± 0.54	0.07 ± 0.66	-0.11 ± 0.98	0.39
Liver stiffness measurement# (KPa)	$-1.30 (-2.70, -0.20)$	$-1.45 (-3.60, 0.40)$	$-0.75 (-1.90, -0.20)$	0.21
Weight (kg)*	-2.60 ± 3.50	-1.40 ± 2.70	-1.80 ± 2.60	0.15
BMI (kg/m^2)*	-0.86 ± 1.18	-0.48 ± 0.92	-0.64 ± 0.95	0.22
Serum AST# (<40 IU/L)	$-15.5 (-35.5, -4.2)$	$-11.8 (-24.0, -3.5)$	$-11.4 (-31.0, -4.0)$	0.46
Serum ALT# (<40 IU/L)	$-39.0 (-70.5, -27.0)$	$-38.2 (-56.0, -11.2)$	$-35.8 (-58.0, -12.0)$	0.3
Serum Triglycerides# (<150 mg/dL)	$-14.2 (-60.0, -18.5)$	$-4.5 (-29.0, 48.5)$	$-13.5 (-126.0, 3.0)$	0.034
Controlled attenuation parameter*	-25.0 ± 39.0	-27.8 ± 42.5	-24.0 ± 44.0	0.89
FIB-4#	$-0.01 (-0.29, 0.10)$	$-0.07 (-0.39, 0.08)$	$-0.09 (-0.35, 0.05)$	0.47
APRI#	$-0.18 (-0.56, -0.05)$	$-0.22 (-0.51, -0.03)$	$-0.24 (-0.46, -0.04)$	0.95

* Parametric data (mean $\pm$ SD)# Non-parametric data

Presents the differences among three groups of clinical, biochemical, and fibrosis-related parameters over 24 weeks. A significant reduction was observed in weight (80.10 ± 12.15 vs. 77.50 ± 12.05 kg; $p<0.001$), BMI (27.72 ± 3.28 vs. 26.86 ± 3.50 kg/m^2 ; $p<0.001$), AST [54.10 (43.0 – 72.0) vs. 35.80 (31.0 – 43.0) IU/L; $p<0.001$], ALT [89.10 (69.0 – 126.0) vs. 47.00 (37.0 – 61.0) IU/L; $p<0.001$], triglycerides [178.90 (145.0 – 241.0) vs. 164.20 (118.0 – 215.0) mg/dL; $p=0.028$], in Saroglitazar group ($n=60$). Even, liver stiffness measurement [6.80 (5.5 – 8.3) vs. 5.30 (4.9 – 6.5) kPa; $p<0.001$], CAP (301.00 ± 36.50 vs. 276.00 ± 43.20 ; $p<0.001$), and APRI [0.70 (0.54 – 1.02) vs. 0.49 (0.36 – 0.62); $p=0.001$]. No significant changes were detected in FIB-4 ($p=0.32$) or NAFLD fibrosis score ($p=0.50$). In the Vitamin E group

($n=60$), significant progress was noted in weight, BMI, AST, ALT, liver stiffness measurement, CAP, FIB-4, and APRI (all $p<0.05$). However, triglyceride levels increased slightly from 180.00 (137.0 – 228.0) to 192.00 (132.0 – 230.0) mg/dL and were not significantly different ($p=0.90$). The NAFLD fibrosis score also remained unchanged ($p=0.50$). In the Lifestyle Intervention group ($n=50$), significant reductions were noticed in weight (76.40 ± 12.00 vs. 74.70 ± 11.85 kg; $p<0.001$), BMI (27.40 ± 3.71 vs. 26.78 ± 3.60 kg/m^2 ; $p<0.001$), AST, ALT, triglycerides [203.30 (140.0 – 293.0) vs. 154.00 (125.0 – 211.0) mg/dL; $p<0.001$], liver stiffness measurement, CAP, FIB-4, and APRI (all $p<0.05$). Similar to the other groups, the NAFLD fibrosis score did not display a significant change ($p=0.42$).

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Table 3. Within-Group Comparison of Outcome Variables at Baseline and 24 Weeks.

Variable	Saroglitazar group (N=60)			Vitamin E group (N=60)			Only Lifestyle group (N=50)		
	Pre	Post	P value	Pre	Post	P value	Pre	Post	P value
Weight (kg)	80.10 ± 12.15	77.50 ± 12.05	<0.001*	75.20 ± 7.40	74.10 ± 7.40	<0.001*	76.40 ± 12.00	74.70 ± 11.85	<0.001*
BMI (kg/m ²)	27.72 ± 3.28	26.86 ± 3.50	<0.001*	26.90 ± 4.20	26.42 ± 3.95	<0.001*	27.40 ± 3.71	26.78 ± 3.60	<0.001*
Serum AST (<40 IU/L)	57.50 ± 21.50	37.40 ± 8.90	<0.001*	57.50 ± 23.00	39.00 ± 16.30	<0.001*	60.00 ± 29.60	41.20 ± 18.50	<0.001*
Serum ALT (<40 IU/L)	97.50 ± 42.20	49.00 ± 17.80	<0.001*	94.00 ± 31.10	58.00 ± 37.00	<0.001*	97.00 ± 53.30	56.00 ± 27.40	<0.001*
Serum Triglycerides (<150 mg/dL)	178.90 ± 71.10	164.20 ± 71.90	0.028*	180.00 ± 67.40	192.00 ± 72.60	0.90*	203.30 ± 113.30	154.00 ± 63.70	<0.001*
Liver Stiffness Measurement (kPa)	6.80 ± 2.07	5.30 ± 1.19	<0.001*	7.10 ± 2.59	5.40 ± 1.78	<0.001*	6.70 ± 2.07	5.80 ± 1.33	<0.001*
Controlled Attenuation Parameter	301.00 ± 36.50	276.00 ± 43.20	<0.001*	297.50 ± 36.30	276.00 ± 41.50	<0.001*	303.80 ± 49.80	277.00 ± 45.50	0.001*
FIB-4	1.02 ± 0.50	0.93 ± 0.39	0.32*	1.19 ± 0.54	1.08 ± 0.50	0.04*	1.29 ± 0.74	0.98 ± 0.47	0.01*
APRI	0.70 ± 0.36	0.49 ± 0.19	0.001*	0.77 ± 0.38	0.60 ± 0.32	<0.001*	0.78 ± 0.42	0.55 ± 0.30	<0.001*
NAFLD Fibrosis Score	-2.82 ± 0.70	-2.75 ± 0.84	0.50*	-2.63 ± 0.92	-2.56 ± 0.90	0.50*	-2.45 ± 1.01	-2.57 ± 1.08	0.42*

Compares the proportion of patients who completed a clinically meaningful reduction of at least 2kPa in liver stiffness after 24 weeks of intervention. The Saroglitazar group presented the highest response rate, with 40%, while the Vitamin E group had 35% and the Only Lifestyle group arm had 20%. There was no statistically significant difference between the Saroglitazar and Vitamin E groups (40% vs. 35%; difference: 5.0%, 95% CI: -13.5 to 23.5; p=0.60), and this study indicates comparable efficacy, which achieved a ≥2 kPa reduction in liver

stiffness. In addition, the difference between the Vitamin E and Lifestyle Intervention groups did not reach statistical significance (35% vs. 20%; difference: 15.0%, 95% CI: -2.4 to 32.4; p=0.089). However, the Saroglitazar group demonstrated a significantly higher proportion of responders than the Only Lifestyle group arm (40% vs. 20%; difference: 20.0%, 95% CI: 2.1 to 37.9; p=0.028). This study suggests that Saroglitazar was more effective than lifestyle change by improving liver stiffness over the 24-week study period.

Table 4. Patients with ≥2 kPa Reduction in Liver Stiffness After 24 Weeks

Comparison	Proportions	Difference in Proportions (95% CI)	P value
Saroglitazar group vs. Vitamin E group	40% vs. 35%	5.0 (-13.5, 23.5)	0.6
Saroglitazar group vs. Only Lifestyle group	40% vs. 20%	20.0 (2.1, 37.9)	0.028
Vitamin E group vs. Only Lifestyle group	35% vs. 20%	15.0 (-2.4, 32.4)	0.089

Figure 1 demonstrates improvements in both fibrosis markers across all treatment groups after 24 weeks. The Lifestyle Modification group showed the greatest reductions in both FIB-4 (-0.09) and

APRI (-0.24), followed closely by the Vitamin E group (FIB-4: -0.07; APRI: -0.22). The Saroglitazar group exhibited the smallest changes (FIB-4: -0.01; APRI: -0.18). Overall, although all inter-

ventions were associated with modest improvements in fibrosis indices, the differences between treatment groups were relatively small, suggesting comparable

efficacy in improving non-invasive fibrosis markers over the study period.

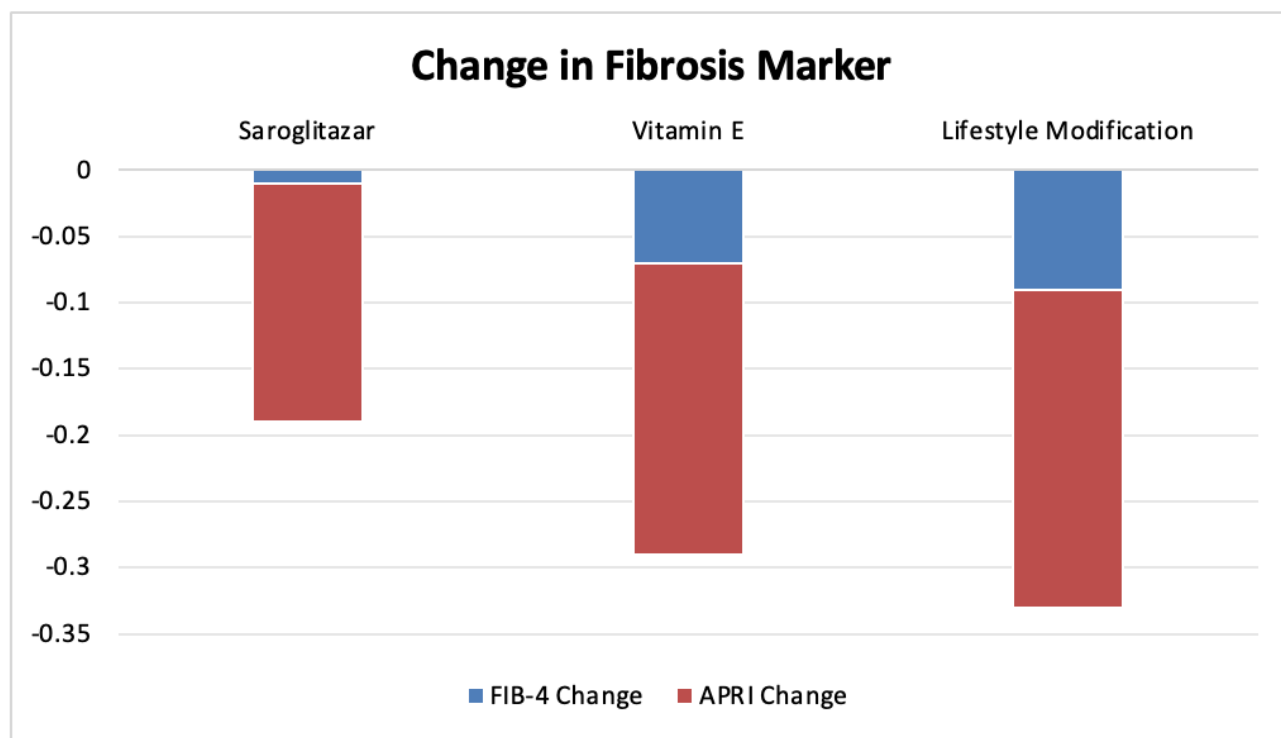


Fig. 1: Change in Markers of Fibrosis in each group

4 | DISCUSSION

The study by Sanyal et al. (2001) evaluated the impact of the saroglitazar, vitamin E, their combination, and lifestyle control among patients with NAFLD/NASH for 24 weeks. Combination therapy provided the greatest benefit, reduces the liver stiffness, hepatic steatosis, liver enzyme levels, insulin resistance and also improves the glycaemic and lipid profiles. Saroglitazar improved the alanine aminotransferase, controlled attenuation parameter (CAP), HbA1c, triglycerides, and LDL cholesterol. The vitamin E reduced the liver enzyme levels with limit impact on the fibrosis and metabolic parameters. The study findings suggested that the combined impact of the saroglitazar and vitamin E therapy is more effective than the monotherapy to improve the hepatic and metabolic outcomes. Consistently our study also demonstrated that Saroglitazar showed high improvement in the level of triglyceride and

the liver stiffness rather than lifestyle changes. While the vitamin E showed improvement impact on the liver enzymes with limited benefit in fibrosis-related outcomes. (13). The impact of saroglitazar, vitamin E, and lifestyle intervention among 150 patients of MASLD showed no differences among the three groups to improve the liver stiffness or the NAFLD fibrosis scores, which indicated that pharmacological therapy did not demonstrate superiority for fibrosis-associated outcomes. Comparable secondary outcomes were observed, except for the reduction in the triglyceride levels with saroglitazar. A high proportion of patients who had received saroglitazar showed a reduction in liver stiffness rather than the lifestyle intervention, showed effective improvement. While our study showed non-significant differences between the groups in liver stiffness or NAFLD fibrosis score, while the Saroglitazar showed the effective reduction in the triglyc-

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eride level and high proportion of patients those who had achieved with liver stiffness improvement rather than lifestyle intervention (14). Similarly, Shah et al. (2025) evaluated the safety and efficacy of the saroglitazar among patients with MASLD/MASH who have completed follow up period. The treatment with Saroglitazar resulted in improvement in the liver biochemistry, lipid profile, glycaemic control and NAFLD fibrosis score, among both of the diabetic and non-diabetic patients. The body mass index showed improvement, which was not statistically significant. No serious adverse effect of the drug was noted. These study findings suggested that saroglitazar is a safe and reliable therapeutic strategy to improve the hepatic, metabolic, and fibrosis-related outcomes among patients. Consistent with Shah et al. (2025) our study also showed better liver enzymes, lipid profile index and fibrosis associated factors, while the level of triglyceride reduced with high response of the liver stiffness. No improvement was noted in the NAFLD fibrosis score (15). Similarly, the study by Gupta et al. (2026), showed improved treatment outcome for the liver stiffness measurement and regulated the attenuation, which indicated the regression of fibrosis and hepatic steatosis. Patients showed reductions in fibrosis stage and steatosis grade. Significant improvements were also noted in fasting blood glucose level, and postprandial blood glucose, HbA1c, alanine aminotransferase, triglycerides, and the triglyceride-glucose index, while some factors remain unchanged, including the BMI, AST, HDL cholesterol, platelet count, and FIB-4 score. The study stated that saroglitazar showed significant hepatic and metabolic improvement apart from alterations in body weight. Our study showed that Saroglitazar improved the liver stiffness, liver enzymes, and triglyceride levels, while the BMI and FIB-4 showed differences in group apart from the improvements in the hepatic and metabolic outcomes (16). The study by Tavaglione F (2024) stated that the trial study compared the impact of saroglitazar, vitamin E, their combination, and lifestyle intervention among patients. Effective therapeutics showed benefit in the liver stiffness, alanine aminotransferase levels, insulin resistance, glycaemic control, and HDL cholesterol rather than the parameters for lifestyle modification. Saroglitazar monotherapy benefited the liver enzyme, HbA1c,

LDL cholesterol, and metabolic parameters, while vitamin E had showed improvement in the liver injury with limited metabolic impact. The study showed a short follow-up period and a lack of liver biopsy, but supported the future investigation of the combined impact of saroglitazar and vitamin E therapy (17).

5 | CONCLUSION

This study concluded that it compared the effects of Saroglitazar, Vitamin E, and lifestyle modification on liver fibrosis markers and metabolic parameters in patients with MASLD/MASH over 24 weeks. Each group presented significant improvements in body weight, BMI, liver enzymes (AST and ALT), liver stiffness measurement, controlled attenuation parameter, and APRI scores. However, no significant between-group differences were found for most fibrosis-related outcomes. Saroglitazar demonstrated a greater reduction in serum triglyceride levels linked with Vitamin E and lifestyle intervention alone. Moreover, a significantly higher proportion of patients in the Saroglitazar group achieved a clinically meaningful reduction of ≥ 2 kPa in liver stiffness compared with the lifestyle intervention group. Furthermore, these progresses, NAFLD fibrosis scores remained largely unchanged across all groups. Moreover, Saroglitazar appears to provide further metabolic and hepatic assistances, mainly in triglyceride reduction and liver stiffness improvement, while Vitamin E and lifestyle modification also contribute to meaningful clinical improvements in MASLD/MASH patients.

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