UKR Journal of Medicine and Medical Research (UKRJMMR)
Homepage: https://ukrpublisher.com/ukrjmmr/
Email: submit.ukrpublisher@gmail.com
Volume 1, Issue 4, Nov-Dec 2025 ISSN: 3107-5061 (Online)

Generic Saroglitazar for Management of Fatty Liver - A Prospective, Observational, Real-World Study from Bangladesh

Ahmed Lutful Moben[1], Rokshana Begum[2], Sheikh Mohammad Noor E Alam[3], Md. Abdur Rahim[4], Sheikh Zahir Raihan[5], Sakirul Khan[6], Md. Rezwanur Rahman[7], Manas Saha[8], Musarrat Mahtab[9], Sheikh Mohammad Fazle Akbar[10], Mamun Al Mahtab[11]

[1]Department of Hepatology, Kurmitola General Hospital, Dhaka, Bangladesh.
[2]Department of Hepatology, Shaheed Suhrawardy Medical College, Dhaka, Bangladesh.
[3]Department of Hepatology, Bangladesh Medical University, Dhaka, Bangladesh.
[4]Department of Hepatology, International Medical College, Gazipur, Bangladesh.
[5]Department of Clinical Pharmacy and Pharmacology, University of Dhaka, Dhaka, Bangladesh.
[6]Research Center for Global and Local Infectious Diseases, Oita University, Yufu, Oita, Japan; Department of Microbiology, Faculty of Medicine, Oita University, Yufu, Oita, Japan; Dhaka, Bangladesh.
[7]National Gastroliver Institute and Hospital, Dhaka, Bangladesh.
[8]Department of Hepatology, Khulna Medical College, Khulna, Bangladesh.
[9]Clinical Research Organization Ltd., Dhaka, Bangladesh.
[10]Department of Gastroenterology and Metabolism, Ehime University Graduate School of Medicine, Ehime, Japan; Research Center for Global and Local Infectious Diseases, Oita University, Yufu, Oita, Japan; Clinical Research Organization Ltd., Dhaka, Bangladesh; Miyakawa Memorial Research Foundation, Tokyo, Japan.
[11]Department of Hepatology, Bangladesh Medical University, Dhaka, Bangladesh.

***Corresponding Author**: Professor Mamun Al Mahtab

DOI: *https://doi.org/10.5281/zenodo.18047012*

Article History
Original Research Article
Received: 09-12-2025
Accepted: 21-12-2025
Published: 24-12-2025

Citation: Ahmed Lutful Moben, Rokshana Begum, Sheikh Mohammad Noor E Alam, Md. Abdur Rahim, Sheikh Zahir Raihan, Sakirul Khan, Md. Rezwanur Rahman, Manas Saha, Musarrat Mahtab, Sheikh Mohammad Fazle Akbar, Prof. Mamun Al Mahtab. (2025), Generic Saroglitazar for Management of Fatty Liver - A Prospective, Observational, Real-World Study from Bangladesh, UKR Journal of Medicine and Medical Research (UKRJMMR), Volume 1(4), 31-36.

Abstract

Introduction: *Fatty liver is a global pandemic and is recognized as one of the most important etiology for liver cirrhosis and hepatocellular carcinoma across the world. While lifestyle modification remains the cornerstone for fatty liver management, drugs are also gradually becoming available to address the issue.*

Materials & Methods: *We recruited 52 patients diagnosed as having fatty liver in this prospective, real-life study. They were randomized into two groups. Saroglitazar group had 30 patients who were prescribed generic saroglitazar 4 mg orally, daily and lifestyle modification. The control group included 20 fatty liver patients, who were advised only lifestyle modification. Patients were followed up at 6 months. Serum alanine aminotransferase and aspartate aminotransferase levels, ultrasonography of hepatobiliary system and Fibroscan were checked at baseline and repeated at 6 months.*

Results: *We observed significant reduction in hepatic fat content as well as improvement in liver enzymes in the saroglitazar group compared to the control group. Reduction in hepatic fibrosis was also seen, but it was not statistically significant.*

Conclusion: *Our real-life study shows promising results with generic saroglitazar in fatty liver patients, justifying the need for larger clinical trials.*

Keywords: *Fatty liver, saroglitazar, generic, liver enzymes, hepatic fat, hepatic fibrosis.*

Introduction

Fatty liver is one of the most common causes of chronic liver disease in the world [1]. The overall global prevalence of fatty liver is 25.2% [2]. Spectrum of fatty liver ranges from fatty liver to liver cirrhosis, liver failure and hepatocellular carcinoma [3, 4]. In Bangladesh, the situation is equally worrisome as the prevalence of fatty liver has been estimated at 18.5% in Bangladesh [5].

Peroxisome proliferator-activated receptor (PPAR) has wide range of metabolic actions that modulate lipid, glucose and energy homeostasis. They also have anti-inflammatory and anti-fibrotic effects [6, 7, 8]. They are primarily seen in liver and adipose tissue and activate fatty acid oxidation [7, 8, 9]. Saroglitazar is a dual PPAR-α/ϒ agonist, which increases hepatic fatty acid oxidation and reduces triglyceride synthesis in the liver [10]. It increases insulin sensitivity and reduces blood glucose and glycosylated hemoglobin (HbA1c) [11]. In high-fat or Western diet-induced steatohepatitis mice model, saroglitazar significantly reduced serum ALT and hepatic fat content as well as hepatic ballooning and lobular inflammation [12, 13].

Material & Methods

In this prospective, observational, real-world study from Bangladesh, we included total 52 treatment naïve, fatty liver patients. The diagnosis of fatty liver was established at ultrasonography of hepatobiliary system and Fibroscan. Hepatitis B and C, history of alcohol intake and liver cirrhosis were excluded. Patients were between 20 to 70 years of age with equal number of 26 males and 26 females. The patients were randomized by block randomization method into 2 groups. In 'saroglitazar group', there were 32 patients. They received saroglitazar 4 mg orally, daily plus lifestyle modification. Generic version of saroglitazar is available in Bangladesh. The "control group' had 20 patients. They were advised lifestyle modification only. Patients were followed up at 6 months to assess response. Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST), ultrasonography of hepatobiliary system and continued attenuation parameter (CAP) and liver stiffness measurement (LSM) Fibroscan were checked.

Results

At baseline, there were no significant differences between the saroglitazar group and the control group in terms of age (44.5 ± 10.6 vs. 41.8 ± 8.8 years, p = 0.34), gender distribution (male 46.9% vs. 55%, p = 0.58), ALT (48.6 ± 40.3 vs. 62.9 ± 40.9 U/L, p = 0.22), AST (35.3 ± 19.5 vs. 45.6 ± 28.4 U/L, p = 0.13) or LSM (6.5 ± 2.8 vs. 5.5 ± 1.0 kPa, p = 0.11). However, CAP values were significantly lower in the saroglitazar group compared to controls at baseline (293.0 ± 33.4 vs. 319.0 ± 34.9 dB/m, p = 0.01) (Tables 1 and 2).

After six months of follow-up, the saroglitazar group showed significantly lower ALT (25.3 ± 14.6 vs. 44.3 ± 22.0 U/L, $p < 0.001$), AST (24.4 ± 6.5 vs. 33.5 ± 9.9 U/L, $p < 0.001$) and CAP values (256.5 ± 37.8 vs. 305.5 ± 33.2 dB/m, $p < 0.001$) compared to controls. No significant difference was, however, observed in LSM between groups (5.1 ± 1.3 vs. 5.4 ± 1.0 kPa, p = 0.41) (Figure 1).

The present study demonstrates that saroglitazar treatment was associated with significant improvements in liver function tests and hepatic steatosis and also showed a favorable reduction in fibrosis over 6 months. While both the saroglitazar and control groups exhibited improvements in serum ALT, AST and CAP values, the magnitude of benefit was substantially greater in the saroglitazar group. Also to be noted is that LSM improved significantly only in the saroglitazar group, suggesting potential antifibrotic effect of the drug.

Discussion

With limited therapeutic options, lifestyle modification and dietary changes remain the main focus of fatty liver management [14, 15]. The fundamentals of lifestyle modification include diet, exercise and weight reduction. Exercise includes brisk walking, aerobic exercise, swimming and cycling [16]. It has been postulated that reduction of >7% body weight leads to resolution of excess hepatic fat, while >10% weight reduction will cause disappearance of hepatic fibrosis [17]. However, despite efforts focusing on lifestyle modifications, preventing long-term complications of metabolic syndrome like fatty liver remains a major challenge [18]. 'Nutrition transition' in the Indian sub-continent, which include obesity, physical inactivity and unhealthy food habit due to socio-economic shift has been crucial in increasing the burden to fatty liver in our part of the globe [19, 20]. Besides, as many patients are unable to maintain dietary and lifestyle changes, long-term outcome of such interventions remains questionable [14, 21]. Rebound weight gain, which is a natural phenomenon, further add to this problem in many cases [22].

Resmetirom, which is a partial activator of a thyroid hormone receptor, reduces hepatic fat was recently approved by the United States Food and Drug Administration (USFDA) as the first drug for the management of liver fat and steatohepatitis [23]. It is also approved by the Directorate General of Drug Administration (DGDA) of Bangladesh. More recently a phase III clinical trial has shown excellent results with semaglutide in liver fat and steatohepatitis management

[24]. Generic saroglitazar, on the other hand, is approved by the DGDA and Central Drugs Standard Control Organization (CDSCO) of India for fatty liver management. It is also produced by several pharmaceuticals in Bangladesh, which is a major hub for manufacturing generic drugs being exported overseas too. We therefore felt that it was important to assess the effectiveness of local generic saroglitazar in fatty liver patients. We checked serum ALT and AST, ultrasonogram of hepatobiliary system and hepatic fat content (*i.e.* CAP) and fibrosis (*i.e.* LSM) by Fibroscan at baseline and then again at 6 months to assess response to generic saroglitazar.

Although, liver biopsy is the 'gold standard' for assessment of hepatic inflammation and fibrosis and our group also performs this procedure routinely, the acceptability of liver biopsy is limited to conservative colleagues as it is an invasive procedure. On the other hand, LSM and CAP by Fibroscan are useful and accepted alternatives for assessment of hepatic fat content and fibrosis [25, 26, 27, 28]. Ultrasonography of hepatobiliary system was not considered for measuring hepatic fat, as it is subject to intra- and inter-observer variability. This is perhaps applicable more for Bangladesh as here this procedure is often performed by non-specialists.

Several recent studies have explored the safety and efficacy of saroglitazar in fatty liver. 101 fatty liver patients out of 107, who were included in a prospective, observational, real-world study after screening 163 patients revealed after 24 weeks that saroglitazar resulted in significant reduction of serum ALT from 94 U/L (47-122 U/L) to 39 U/L (31-49 U/L) ($p<0.0001$) and serum AST 89 U/L (43-114 U/L) to 37 U/L (30-47 U/L) ($p<0.0001$). LSM also reduced from 8.4 kPa (7.1-9.3 kPa) to 7.5 kPa (6.4-8.4 kPa) ($p = 0.0261$). CAP also improved significantly [3]. Another randomized, controlled, double blind, phase-II trial showed that compared to placebo at 16 weeks, saroglitazar (4 mg/day, orally) improved liver fat content (4.1% [5.9] vs -19.7% [5.6]) ($p<0.05$) and serum ALT -45.8% vs 3.4% ($p<0.001$) [29]. This study had 106 fatty liver patients. A recent and much larger retrospective, observational, electronic medical record-based, real-world study involving 553 fatty liver patients revealed that at 90 days, saroglitazar (4 mg/day, orally) resulted in improved hepatic parameters with serum ALT and AST decreasing by -2.62 U/L and -7.95 U/L respectively [30]. A more recent retrospective, single center, observational study included 204 fatty liver patients who received oral saroglitazar 4 mg, once daily. The study observed that in significant fibrosis group (<14 kPa), LSM reduced from 10.31±2.01 kPa to 6.27±1.44 kPa (-39.1%, $p<0.001$) and CAP from 295.82±49.34 dB/m to 241.01±63.61 dB/m (-18.4%). ALT and AST declined by 49.5% and 43.4% respectively. In the advanced fibrosis group (>14 kPa), LSM decreased from 17.96±1.85 kPa to 13.83±1.42 kPa (-23.0%) and CAP from 31.05±61.28 dB/m to 272.36±52.38 dB/m (-14.1%). ALT and AST reductions were 46.6% and 45.1% respectively [31]. In all these studies saroglitazar was well tolerated. Adverse events of saroglitazar included pyrexia, dyspepsia, gastritis and asthenia [30, 32, 3].

The findings align with earlier reports indicating that dual PPAR-α/γ agonists exert beneficial effects on dysregulated lipid metabolism and hepatic inflammation, contributing to improvements in fatty liver and steatohepatitis. The significant reduction in CAP highlights saroglitazar's efficacy in ameliorating hepatic steatosis, while the parallel reduction in liver enzymes suggests an improvement in hepatocellular injury. The modest, but significant decline in LSM in the treatment group raises the possibility that longer-term therapy could further impact fibrosis progression, an aspect not observed in the control cohort.

This study has some limitations, including the relatively small sample size and short follow-up duration. Nonetheless, the consistent improvements across biochemical and imaging markers provide encouraging evidence supporting saroglitazar's role in fatty liver and/or steatohepatitis management. Future randomized controlled trials with larger cohorts and extended follow-up are warranted to confirm these findings and evaluate long-term histological outcomes. This real-life observation is also important as it also demonstrates efficacy of Bangladeshi generic saroglitazar in fatty liver.

Conclusion

In Bangladesh, like many other countries of the region, we are faced with pandemic of fatty liver. It is further anticipated that fatty liver will pose larger burden to the health system of the country in the future. It is therefore critical for us to explore different approaches, in addition to lifestyle modification, for management of fatty liver and steatohepatitis. Although, this is a small real-life study, it yields promising data justifying the need for multi-center clinical trial in fatty liver with generic saroglitazar.

References

1. Duseja A, Singh SP, Saraswat VA, Acharya SK, Chawla YK, Chowdhury S, Dhiman RK, Jayakumar RV, Madan K, Misra SP, Mishra H, Modi SK, Muruganathan A, Saboo B, Sahay R, Upadhyay R. Non-alcoholic Fatty Liver Disease and Metabolic Syndrome-Position Paper of the Indian National Association for the Study of the Liver, Endocrine Society of India, Indian College of Cardiology and Indian Society of Gastroenterology. J Clin Exp Hepatol. 2015 Mar;5(1):51-68. doi:

10.1016/j.jceh.2015.02.006. Epub 2015 Mar 6. PMID: 25941433; PMCID: PMC4415196.

2. Chalasani Naga, Younossi Zobair, Lavine Joel E, Charlton Michael, Cusi Kenneth, Rinella Mary, Harrison Stephen A, Brunt Elizabeth M, Sanyal Arun J. The diagnosis and management of nonalcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. Hepatology 67(1):p 328-357, January 2018. DOI: 10.1002/hep.29367.
3. Goyal O, Nohria S, Goyal P, Kaur J, Sharma S, Sood A, Chhina RS. Saroglitazar in patients with non-alcoholic fatty liver disease and diabetic dyslipidemia: a prospective, observational, real world study. Sci Rep. 2020 Dec 3;10(1):21117. doi: 10.1038/s41598-020-78342-x. PMID: 33273703; PMCID: PMC7713236.
4. Cotter TG, Rinella M. Nonalcoholic Fatty Liver Disease 2020: The State of the Disease. Gastroenterology. 2020 May;158(7):1851-1864. doi: 10.1053/j.gastro.2020.01.052. Epub 2020 Feb 13. PMID: 32061595.
5. Rahman MM, Kibria MG, Begum H, Haque M, Sultana N, Akhter M, Rowshon AHM, Ahmed F, Hasan M. Prevalence, risk factors and metabolic profile of the non-obese and obese non-alcoholic fatty liver disease in a rural community of South Asia. BMJ Open Gastroenterol. 2020 Dec;7(1):e000535. doi: 10.1136/bmjgast-2020-000535. PMID: 33376110; PMCID: PMC7778747.
6. Francque S, Szabo G, Abdelmalek MF, Byrne CD, Cusi K, Dufour JF, Roden M, Sacks F, Tacke F. Nonalcoholic steatohepatitis: the role of peroxisome proliferator-activated receptors. Nat Rev Gastroenterol Hepatol. 2021 Jan;18(1):24-39. doi: 10.1038/s41575-020-00366-5. Epub 2020 Oct 22. PMID: 33093663.
7. Poulsen Ll, Siersbæk M, Mandrup S. PPARs: fatty acid sensors controlling metabolism. Semin Cell Dev Biol. 2012 Aug;23(6):631-9. doi: 10.1016/j.semcdb.2012.01.003. Epub 2012 Jan 18. PMID: 22273692.
8. Polyzos SA, Kang ES, Boutari C, Rhee EJ, Mantzoros CS. Current and emerging pharmacological options for the treatment of nonalcoholic steatohepatitis. Metabolism. 2020 Oct;111S:154203. doi: 10.1016/j.metabol.2020.154203. Epub 2020 Mar 6. PMID: 32151660.
9. Wang Y, Nakajima T, Gonzalez FJ, Tanaka N. PPARs as Metabolic Regulators in the Liver: Lessons from Liver-Specific PPAR-Null Mice. Int J Mol Sci. 2020 Mar 17;21(6):2061. doi: 10.3390/ijms21062061. PMID: 32192216; PMCID: PMC7139552.
10. Jani RH, Kansagra K, Jain MR, Patel H. Pharmacokinetics, safety, and tolerability of saroglitazar (ZYH1), a predominantly PPARα agonist with moderate PPARγ agonist activity in healthy human subjects. Clin Drug Investig. 2013 Nov;33(11):809-16. doi: 10.1007/s40261-013-0128-3. PMID: 24062180; PMCID: PMC3824340.
11. Pai V, Paneerselvam A, Mukhopadhyay S, Bhansali A, Kamath D, Shankar V, Gambhire D, Jani RH, Joshi S, Patel P. A Multicenter, Prospective, Randomized, Double-blind Study to Evaluate the Safety and Efficacy of Saroglitazar 2 and 4 mg Compared to Pioglitazone 45 mg in Diabetic Dyslipidemia (PRESS V). J Diabetes Sci Technol. 2014 Jan;8(1):132-141. doi: 10.1177/1932296813518680. Epub 2014 Jan 16. PMID: 24876549; PMCID: PMC4390522.
12. Jain MR, Giri SR, Bhoi B, Trivedi C, Rath A, Rathod R, Ranvir R, Kadam S, Patel H, Swain P, Roy SS, Das N, Karmakar E, Wahli W, Patel PR. Dual PPARα/γ agonist saroglitazar improves liver histopathology and biochemistry in experimental NASH models. Liver Int. 2018 Jun;38(6):1084-1094. doi: 10.1111/liv.13634. Epub 2017 Dec 14. PMID: 29164820; PMCID: PMC6001453.
13. Kumar DP, Caffrey R, Marioneaux J, Santhekadur PK, Bhat M, Alonso C, Koduru SV, Philip B, Jain MR, Giri SR, Bedossa P, Sanyal AJ. The PPAR α/γ Agonist Saroglitazar Improves Insulin Resistance and Steatohepatitis in a Diet Induced Animal Model of Nonalcoholic Fatty Liver Disease. Sci Rep. 2020 Jun 9;10(1):9330. doi: 10.1038/s41598-020-66458-z. PMID: 32518275; PMCID: PMC7283326.
14. Bellentani S, Dalle Grave R, Suppini A, Marchesini G; Fatty Liver Italian Network. Behavior therapy for nonalcoholic fatty liver disease: The need for a multidisciplinary approach. Hepatology. 2008 Feb;47(2):746-54. doi: 10.1002/hep.22009. PMID: 18098321.
15. Chalasani N, Younossi Z, Lavine JE, Diehl AM, Brunt EM, Cusi K, Charlton M, Sanyal AJ; American Gastroenterological Association; American Association for the Study of Liver Diseases; American College of Gastroenterology. The diagnosis and management of non-alcoholic fatty liver disease: practice guideline by the American Gastroenterological Association, American Association for the Study of Liver Diseases, and American College of Gastroenterology. Gastroenterology. 2012 Jun;142(7):1592-609. doi: 10.1053/j.gastro.2012.04.001. Epub 2012 May 15.

Erratum in: Gastroenterology. 2012 Aug;143(2):503. PMID: 22656328.
16. Marchesini G, Day CP, Dufour JF, et al. EASL-EASD-EASO Clinical Practice Guidelines for the management of nonalcoholic fatty liver disease. J Hepatol 2016;64(6):1388–1402. DOI: 10.1016/j.jhep.2015.11.004.
17. Hannah W, Harrison S. Effect of weight loss, diet, exercise, and bariatric surgery on nonalcoholic fatty liver disease. Clin Liver Dis 2016;20(2):339–350. Available from: https://pubmed.ncbi.nlm.nih.gov/27063273/.
18. Lillich FF, Imig JD, Proschak E. Multi-Target Approaches in Metabolic Syndrome. Front Pharmacol. 2021 Mar 12;11:554961. doi: 10.3389/fphar.2020.554961. PMID: 33776749; PMCID: PMC7994619.
19. Bhalwar R. Metabolic syndrome: The Indian public health perspective. Med J Armed Forces India. 2020 Jan;76(1):8-16. doi: 10.1016/j.mjafi.2019.12.001. Epub 2020 Jan 13. PMID: 32020962; PMCID: PMC6994803.
20. Tackes N. Metabolic living: Food, fat, and the absorption of illness in India. Glob Public Health. 2018 Aug 18:1-2. doi: 10.1080/17441692.2018.1511742. Epub ahead of print. PMID: 30122098.
21. Musso G, Gambino R, Cassader M, Pagano G. A meta-analysis of randomized trials for the treatment of nonalcoholic fatty liver disease. Hepatology. 2010 Jul;52(1):79-104. doi: 10.1002/hep.23623. PMID: 20578268.
22. Roy PP, Mahtab MA, Rahim MA, Yesmin SS, Islam SB, Akbar SMF. Treatment of Nonalcoholic Steatohepatitis by Obeticholic Acid: Current Status. Euroasian J Hepatogastroenterol. 2022 Jul;12(Suppl 1):S46-S50. doi: 10.5005/jp-journals-10018-1360. PMID: 36466097; PMCID: PMC9681570.
23. https://www.fda.gov/news-events/press-announcements/fda-approves-first-treatment-patients-liver-scarring-due-fatty-liver-disease.
24. Sanyal AJ, Newsome PN, Kliers I, Østergaard LH, Long MT, Kjær MS, Cali AMG, Bugianesi E, Rinella ME, Roden M, Ratziu V; ESSENCE Study Group. Phase 3 Trial of Semaglutide in Metabolic Dysfunction-Associated Steatohepatitis. N Engl J Med. 2025 Apr 30. doi: 10.1056/NEJMoa2413258. Epub ahead of print. PMID: 40305708.
25. Foucher J, Chanteloup E, Vergniol J, Castéra L, Le Bail B, Adhoute X, Bertet J, Couzigou P, de Lédinghen V. Diagnosis of cirrhosis by transient elastography (FibroScan): a prospective study. Gut. 2006 Mar;55(3):403-8. doi: 10.1136/gut.2005.069153. Epub 2005 Jul 14. PMID: 16020491; PMCID: PMC1856085.
26. Wong VW, Vergniol J, Wong GL, Foucher J, Chan HL, Le Bail B, Choi PC, Kowo M, Chan AW, Merrouche W, Sung JJ, de Lédinghen V. Diagnosis of fibrosis and cirrhosis using liver stiffness measurement in nonalcoholic fatty liver disease. Hepatology. 2010 Feb;51(2):454-62. doi: 10.1002/hep.23312. PMID: 20101745.
27. Pu K, Wang Y, Bai S, Wei H, Zhou Y, Fan J, Qiao L. Diagnostic accuracy of controlled attenuation parameter (CAP) as a non-invasive test for steatosis in suspected non-alcoholic fatty liver disease: a systematic review and meta-analysis. BMC Gastroenterol. 2019 Apr 8;19(1):51. doi: 10.1186/s12876-019-0961-9. PMID: 30961539; PMCID: PMC6454693.
28. Chan WK, Nik Mustapha NR, Mahadeva S. Controlled attenuation parameter for the detection and quantification of hepatic steatosis in nonalcoholic fatty liver disease. J Gastroenterol Hepatol. 2014;29(7):1470-6. doi: 10.1111/jgh.12557. PMID: 24548002.
29. Gawrieh S, Noureddin M, Loo N, Mohseni R, Awasty V, Cusi K, Kowdley KV, Lai M, Schiff E, Parmar D, Patel P, Chalasani N. Saroglitazar, a PPAR-α/γ Agonist, for Treatment of NAFLD: A Randomized Controlled Double-Blind Phase 2 Trial. Hepatology. 2021 Oct;74(4):1809-1824. doi: 10.1002/hep.31843. Epub 2021 Jul 19. PMID: 33811367.
30. Das S, Gupta S, Lathia T, Swain J, Mittal S, Kumar S, Dm M, Garg N, Teja R, Beatrice A, Sooragonda BG, Mohd Razi S, Jaiswal A, Jhaveri KS, Shah S, Verma G. Evaluation of Effectiveness and Tolerability of Saroglitazar in Metabolic Disease Patients of India: A Retrospective, Observational, Electronic Medical Record-Based Real-World Evidence Study. Cureus. 2025 Jul 30;17(7):e89028. doi: 10.7759/cureus.89028. PMID: 40896040; PMCID: PMC12395119.
31. Kak M C. Role of Saroglitazar in Improving Transient Elastography Parameters in Significant and Advanced Metabolic Dysfunction-Associated Steatohepatitis. Cureus 2025;17(12):e99044. doi:10.7759/cureus.99044
32. Jani RH, Pai V, Jha P, Jariwala G, Mukhopadhyay S, Bhansali A, Joshi S. A multicenter, prospective, randomized, double-blind study to evaluate the safety and efficacy of Saroglitazar 2 and 4 mg compared with placebo in type 2 diabetes mellitus patients having hypertriglyceridemia not controlled with atorvastatin therapy (PRESS VI). Diabetes Technol Ther. 2014 Feb;16(2):63-71. doi: 10.1089/dia.2013.0253. Epub 2013 Oct 18. PMID: 24138536; PMCID: PMC3894674.

Table 1: Age Distribution of Patients in Saroglitazar and Control group (N =52)

Age group (years)	Saroglitazar (n=32)	Control (n=20)	p-value
21-30	4	1	0.341
31-40	7	8	
41-50	13	7	
51-60	6	3	
61-70	2	1	
Total	32	20	
Mean ± SD	44.53± 10.6	41.80± 8.82	

*p-value obtained by Chi-square test, p<0.05 was considered as a level of *significant*

Table 2: Sex Distribution of Patients in Saroglitazar and Control group (N = 52)

Sex	Saroglitazar	Control	p-value
Male	15	11	0.577
Female	17	9	
Total	32	20	

*p-value obtained by Chi-square test, p<0.05 was considered as a level of *significant*

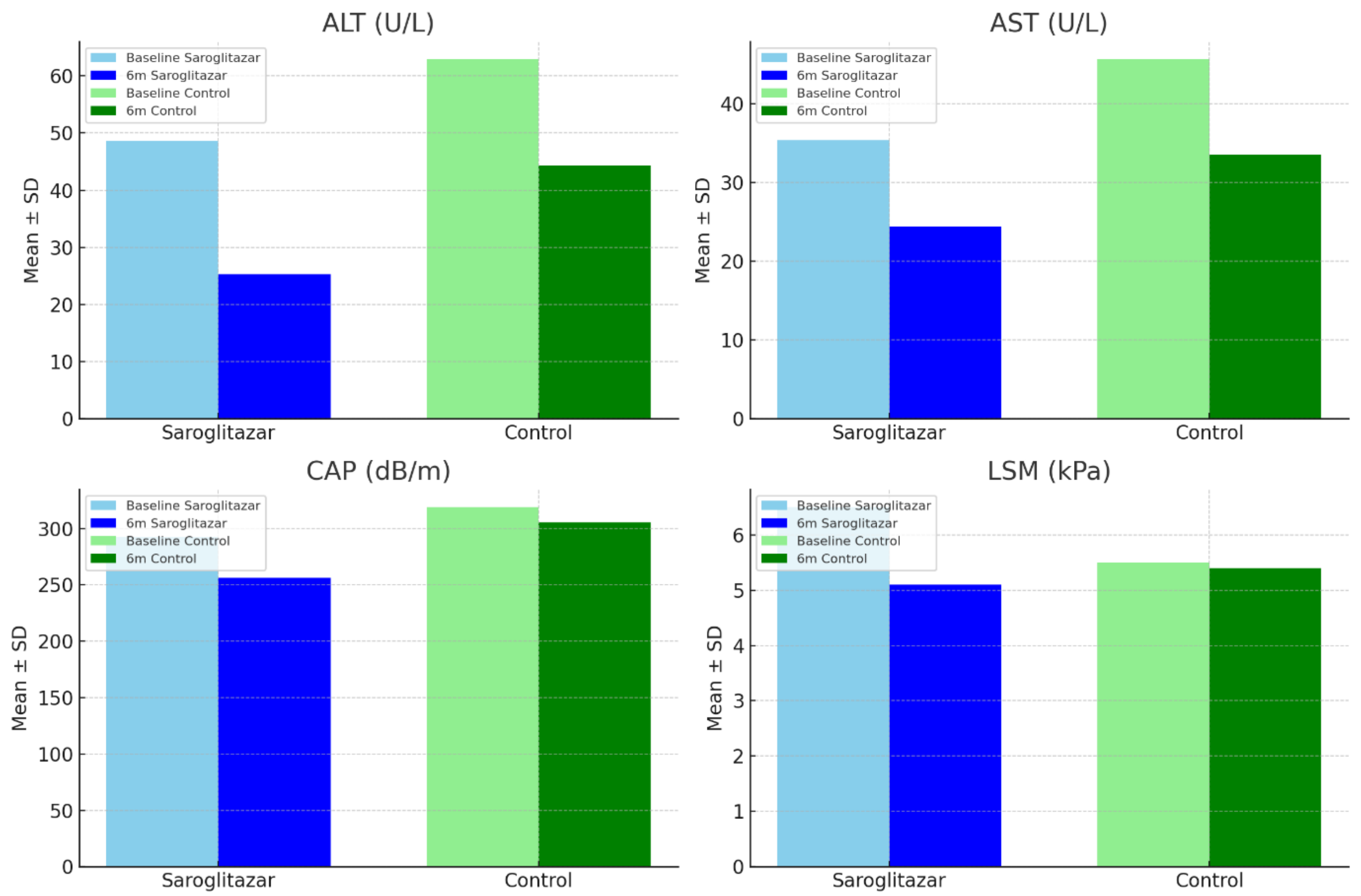

Figure-1: *Within-Group Comparison of ALT, AST, Liver Stiffness (LSM), and Steatosis (CAP) at Baseline and After 6-12 Months in Saroglitazar Group and Control Group (N=52).*