

EFFECT OF STRUCTURED NUTRITION AND LIFESTYLE INTERVENTION ON METABOLIC, HEPATIC, AND ANTHROPOMETRIC OUTCOMES IN TYPE 2 DIABETES MELLITUS PATIENTS WITH AND WITHOUT METABOLIC DYSFUNCTION-ASSOCIATED FATTY LIVER DISEASE: A STRATIFIED RANDOMIZED CONTROLLED TRIAL

Anmayee Nanda^{1*}, Dr Sambit Das², Dr Suryamani Patro³

¹Phd research scholar, Department of Home science, Ramadevi Women's university, ORCID ID: 0009-0008-1541-3605, E-mail ID: anmayeenanda@gmail.com

²MD(Medicine), DM (Endocrinology, PGIMER, Chandigarh),FACE, Professor & Head, Department of Endocrinology, KIMS Medical College and Super speciality Hospital, Bhubaneswar, Senior consultant Endocrinologist Endeavour Clinic, Endocrinology, Orcid ID: 0000-0001-5983-0453, E-mail ID: drsambitendocrine@gmail.com

³Associate Professor, Department of Home science, Ramadevi Women's University, E-mail ID: suryamanipatro@rdwu.ac.in, ORCID ID: 0009-0001-1334-1926

ABSTRACT

Background: Both type 2 diabetes mellitus (T2DM) and metabolic dysfunction-associated fatty liver disease (MAFLD) usually occur parallelly and share a common pathophysiological mechanism. This may include insulin resistance, dyslipidemia, and inflammation. Change in nutrition and lifestyle can help manage these conditions in a cost-effective manner.

Objectives: The aim was to evaluate the effect of dietary and lifestyle intervention in patients with T2DM and MAFLD. To observe the difference in parameters like glycemic, lipid, and anthropometric profiles of subjects over the period of six months.

Methods: A stratified randomized controlled trial was conducted among 300 adults with T2DM from diabetic and gastroenterology clinics in Bhubaneswar, India. Participants were categorized on the basis of the presence of MAFLD. They were randomly divided into a control group and an intervention group. Four groups with 75 participants each were created. The outcomes were measured at different intervals. It was measured at the time of start of the study and then at 1 month, 3 month, and 6 months.

Results: The results were observed across all parameters in the total population. The results showed improvements over six months ($p < 0.001$). Reduction in FBS was observed in MAFLD group. Other parameters reduced were PPBS (34.35 mg/dL), HbA1c (0.91%), ALT (21.80 U/L), AST (17.55 U/L), triglycerides (35.69 mg/dL), BMI (1.17 kg/m²), and weight (3.44 kg), with HDL rising by 4.18 mg/dL. Non-MAFLD intervention group also showed improvements, but control groups experienced only marginal changes. Intervention vs. control comparisons revealed very large effect sizes (Cohen's d : -5.05 to -5.86), with all mean differences significant at $p < 0.001$ and 95% confidence intervals excluding zero.

Conclusion: Significant improvements can be achieved with structured nutrition and lifestyle intervention in various metabolic parameters in T2DM patients. This makes a cornerstone in MAFLD and T2DM management.

KEYWORDS: Dietary counselling, glycemic control, lifestyle intervention, liver enzymes, MAFLD, metabolic syndrome, randomized controlled trial, type 2 diabetes mellitus.

INTRODUCTION

Metabolic dysfunction-associated fatty liver disease (MAFLD) is one of the most prevalent chronic liver conditions. The presence of hepatic steatosis in association with other metabolic risk factors defines the disease. It is also associated with other risk factors like obesity, type 2 diabetes mellitus (T2DM) or sometimes any other metabolic dysregulation [1]. MAFLD and T2DM usually exists parallelly. They also share pathophysiological mechanism like insulin resistance, dyslipidemia, and chronic low-grade inflammation. This creates a bidirectional relationship in which one condition exacerbates the other. [2-3]

India has a much higher burden of T2DM among other countries. The prevalence is rising rapidly due to urbanization, sedentary lifestyle, and dietary transitions. Since MAFLD and T2DM coexists, it flares up the risk of hepatic fibrosis, cardiovascular disease, and all-cause mortality. [4] Structured nutrition therapy and lifestyle modifications are emerging as primary interventions, which are capable of targeting multiple metabolic pathways. [5]

Although there are well-established theories, rationales, and clinical evidence regarding the impact of dietary and lifestyle modifications in MAFLD and non-MAFLD subgroups of T2DM patients, its implementation in India is still limited. The

study is designed to evaluate the effect of structured dietary and lifestyle interventions on a comprehensive panel of glycemic, hepatic, lipid, and anthropometric parameters in T2DM patients over a period of six months.

MATERIALS AND METHODS

Declaration of Helsinki (revised 2000) was followed. The study was not prospectively registered in a clinical trial registry. Informed consent in written was taken from all participants. The confidentiality was maintained throughout the study.

A stratified, randomized controlled trial was conducted across eight different centres in Bhubaneswar, Odisha. The study was carried out in two phases. Phase one included the establishment of a cross-sectional assessment for MAFLD cases, and phase two included a six-month intervention trial. This was followed by follow-up at 1, 3, and 6 months.

Participants who were included in the study were aged between 20 and 70 years with confirmed T2DM for more than one-year. Clinical criteria, biochemical criteria, and ultrasonography were considered to confirm MAFLD in the patients. Type 1 diabetes and other endocrine disorders were excluded from the study. Viral hepatitis, alcohol-related liver disease was also excluded. Individuals with pregnancy, lactation, or any severe systemic illness were also excluded from the study. Another exclusion criterion was the inability to provide informed consent.

Three hundred participants were stratified by MAFLD status (MAFLD, n=150; non-MAFLD, n=150) and randomized in a 1:1 ratio into intervention and control arms, yielding four groups (n=75 each): (1) T2DM+MAFLD+Intervention, (2) T2DM+MAFLD+Control, (3) T2DM without MAFLD + Intervention, and (4) T2DM without MAFLD + Control. Randomization was performed by an independent investigator using a computer-generated random allocation sequence to ensure equal distribution among groups. Due to the nature of the nutrition and lifestyle intervention, participant blinding was not feasible. However, biochemical analyses were conducted at NABL-accredited laboratories using standardized procedures to minimize assessment bias.

Participants from the intervention group were provided with a six-month structured nutrition-therapy program. Personalized meal plans and lifestyle counselling were included in the program. It focused on portion control, dietary quality, and physical activity. Participants' adaptability was monitored via telephone consultations or WhatsApp reminders, and bimonthly visits.

Assessment was done at the time of intervention, and follow-up was done at 1, 3, and 6 months. Biochemical analysis was carried out at NABL-accredited labs. Parameters analyzed were glycemic parameters (FBS, PPBS, HbA1c), hepatic enzymes (ALT, AST, ALP), and anthropometrics (BMI, waist circumference, body weight). The secondary outcomes included blood pressure (SBP, DBP) and lipid profile (triglycerides, HDL).

The statistical Package for the Social Sciences (SPSS) was used to analyze the data. Descriptive statistics are reported as means. Within-group changes were assessed by repeated-measures analysis; between-group comparisons used one-way ANOVA or Kruskal-Wallis test as appropriate. Intervention vs. control comparisons used the independent Welch t-test. Statistical significance was reported as set at $p < 0.05$, with exact p-values reported throughout.

RESULTS

Table 1 here represents the baseline demographic details and clinical characteristics of the four study groups. Table 2 represents the improvement in all clinical and biochemical parameters. These results were the outcome of over a period of six months ($p < 0.001$). The values that were decreased were SBP, DBP, ALT, AST, ALP, and triglycerides. The values that were improved included FBS, PPBS, HbA1c. A significant reduction in BMI, waist circumference, and body weight was noted. A significant improvement across all the parameters was observed in the MAFLD intervention group. It is represented by Table 3. There was a marked reduction in BP, glycemic indices, liver enzymes, triglycerides. The group showed a significant reduction in BMI, waist circumference, and body weight too. It also showed a rise in HDL levels.

Table 1: Baseline Demographic details and Clinical Characteristics of the Four Study Groups

Variable	Non-MAFLD Control (n=75)	Non-MAFLD Intervention (n=75)	MAFLD Control (n=75)	MAFLD Intervention (n=75)
Age group, n (%)				
20–40 years	36 (48.0)	37 (49.3)	30 (40.0)	30 (40.0)
41–60 years	33 (44.0)	33 (44.0)	33 (44.0)	33 (44.0)
>60 years	6 (8.0)	5 (6.7)	12 (16.0)	12 (16.0)
Sex, n (%)				
Male	17 (22.7)	17 (22.7)	28 (37.3)	28 (37.3)
Female	58 (77.3)	58 (77.3)	47 (62.7)	47 (62.7)
Duration of diabetes, n (%)				
≤1 year	14 (18.7)	13 (17.3)	10 (13.3)	10 (13.3)
1–5 years	34 (45.3)	35 (46.7)	38 (50.7)	39 (52.0)
5–10 years	16 (21.3)	16 (21.3)	19 (25.3)	19 (25.3)
>10 years	11 (14.7)	11 (14.7)	8 (10.7)	7 (9.3)
Treatment modality, n (%)				
Oral hypoglycemic				

agents (OHA)	43 (57.3)	43 (57.3)	44 (58.7)	44 (58.7)
Insulin therapy	20 (26.7)	19 (25.3)	20 (26.7)	19 (25.3)

Table 2: Changes in Clinical and Biochemical Parameters Over 6 Months in Total Study Population (N=300)

Variable	Pre	1 Month	3 Month	6 Month	Total Change (Pre-6M)	p-value
SBP (mmHg)	126.63	124.35	121.84	120.40	6.23	<0.001
DBP (mmHg)	81.76	80.76	79.82	79.27	2.49	<0.001
FBS (mg/dL)	181.84	176.61	171.54	166.78	15.06	<0.001
PPBS (mg/dL)	270.89	263.16	256.04	249.31	21.58	<0.001
HbA1c (%)	9.49	9.28	9.10	8.94	0.55	<0.001
ALT (U/L)	81.84	78.01	73.91	70.79	11.05	<0.001
AST (U/L)	72.25	68.83	65.25	62.71	9.54	<0.001
ALP (U/L)	153.47	150.95	148.14	146.09	7.38	<0.001
Triglycerides (mg/dL)	281.48	274.52	267.59	261.89	19.58	<0.001
HDL (mg/dL)	34.92	35.87	36.63	37.47	2.55	<0.001
BMI (kg/m ²)	30.26	30.01	29.77	29.54	0.72	<0.001
Waist Circumference (cm)	100.72	100.08	99.52	98.90	1.82	<0.001
Weight (kg)	82.67	81.99	81.22	80.55	2.12	<0.001

Table 3: Changes in Clinical and Biochemical Parameters Over 6 Months – MAFLD Intervention Group (N=75)

Variable	Pre	1 Month	3 Month	6 Month	Total Change (Pre-6M)	p-value
SBP (mmHg)	128.32	124.97	121.13	118.68	9.64	<0.001
DBP (mmHg)	82.32	81.08	79.44	78.24	4.08	<0.001
FBS (mg/dL)	191.35	182.09	174.46	166.15	25.20	<0.001
PPBS (mg/dL)	291.60	279.28	267.60	257.25	34.35	<0.001
HbA1c (%)	9.92	9.60	9.28	9.01	0.91	<0.001
ALT (U/L)	93.49	86.33	78.05	71.70	21.80	<0.001
AST (U/L)	83.40	77.15	70.59	65.85	17.55	<0.001
ALP (U/L)	161.52	156.41	151.98	147.61	13.91	<0.001
Triglycerides (mg/dL)	300.20	287.63	275.06	264.51	35.69	<0.001
HDL (mg/dL)	33.20	34.58	35.97	37.38	4.18	<0.001
BMI (kg/m ²)	31.37	30.99	30.55	30.20	1.17	<0.001
Waist Circumference (cm)	104.96	103.89	102.79	101.87	3.09	<0.001
Weight (kg)	85.20	83.95	82.69	81.76	3.44	<0.001

Only a minimal improvement was observed in the MAFLD control group over six months. It is represented by Table 4. There was a reduction in BP, glycemic parameters, liver enzymes, and triglycerides, but to a minimal extent. A relatively lower reduction in BMI and body weight, with a slight increase in HDL, was also observed. The clinical impact of these changes was substantially very low as compared to that of the intervention group.

Table 4: Changes in Clinical and Biochemical Parameters Over 6 Months – MAFLD Control Group (N=75)

Variable	Pre	1 Month	3 Month	6 Month	Total Change (Pre–6M)	p-value
SBP (mmHg)	126.73	125.95	124.27	124.15	2.59	<0.001
DBP (mmHg)	82.47	81.49	81.79	81.60	0.87	0.01
FBS (mg/dL)	194.88	193.52	191.34	189.54	5.34	<0.001
PPBS (mg/dL)	294.89	290.94	288.89	286.49	8.40	<0.001
HbA1c (%)	9.91	9.84	9.77	9.71	0.20	<0.001
ALT (U/L)	91.76	90.90	89.76	89.39	2.37	<0.001
AST (U/L)	84.97	83.92	82.83	82.13	2.84	<0.001
ALP (U/L)	167.59	167.00	166.60	165.71	1.88	<0.001
Triglycerides (mg/dL)	306.64	304.68	302.11	299.92	6.72	<0.001
HDL (mg/dL)	32.13	32.37	32.87	33.12	0.99	<0.001
BMI (kg/m ²)	31.95	31.84	31.79	31.68	0.27	<0.001
Waist Circumference (cm)	103.32	103.08	102.94	102.66	0.66	<0.001
Weight (kg)	87.57	87.44	87.22	86.88	0.69	<0.001

A significant improvement in all the parameters was observed in the non-MAFLD intervention group over six months ($p<0.001$). It is clearly represented in Table 5. There was a reduction in BP, glycemic markers, liver enzymes, and triglycerides along with reduction in BMI, waist circumference, and body weight. HDL levels increased significantly.

Table 5: Changes in Clinical and Biochemical Parameters Over 6 Months – Non-MAFLD Intervention Group (N=75)

Variable	Pre	1 Month	3 Month	6 Month	Total Change (Pre–6M)	p-value
SBP (mmHg)	126.63	122.59	119.51	116.89	9.73	<0.001
DBP (mmHg)	81.88	80.20	78.73	78.13	3.75	<0.001
FBS (mg/dL)	170.21	162.46	152.35	145.62	24.59	<0.001
PPBS (mg/dL)	246.89	234.01	223.29	211.89	35.00	<0.001
HbA1c (%)	9.04	8.73	8.41	8.15	0.90	<0.001
ALT (U/L)	70.56	64.17	58.40	53.06	17.50	<0.001
AST (U/L)	60.67	55.48	49.74	45.63	15.04	<0.001
ALP (U/L)	142.36	138.47	133.54	130.42	11.94	<0.001
Triglycerides (mg/dL)	258.16	247.35	236.53	228.52	29.64	<0.001
HDL (mg/dL)	37.55	39.18	40.21	41.55	4.00	<0.001

Variable	Pre	1 Month	3 Month	6 Month	Total Change (Pre–6M)	p-value
BMI (kg/m ²)	28.76	28.35	27.94	27.58	1.18	<0.001
Waist Circumference (cm)	96.90	95.93	94.96	93.93	2.97	<0.001
Weight (kg)	77.41	76.20	74.99	73.84	3.58	<0.001

Only minor changes were found in the non-MAFLD control group, which are represented by Table 6. Minimal improvements were observed in glycemic, hepatic, lipid, and anthropometric parameters. This itself indicates the limited benefit with standard care alone.

Table 6: Changes in Clinical and Biochemical Parameters Over 6 Months – Non-MAFLD Control Group (N=75)

Variable	Pre	1 Month	3 Month	6 Month	Total Change (Pre–6M)	p-value
SBP (mmHg)	124.84	123.91	122.44	121.88	2.96	<0.001
DBP (mmHg)	80.37	80.27	79.32	79.12	1.25	<0.001
FBS (mg/dL)	170.92	168.37	168.01	165.81	5.11	<0.001
PPBS (mg/dL)	250.19	248.39	244.40	241.60	8.58	<0.001
HbA1c (%)	9.07	8.96	8.95	8.87	0.20	<0.001
ALT (U/L)	71.55	70.66	69.45	69.03	2.52	<0.001
AST (U/L)	59.96	58.77	57.82	57.22	2.74	<0.001
ALP (U/L)	142.41	141.94	140.46	140.64	1.77	<0.001
Triglycerides (mg/dL)	260.91	258.41	256.64	254.62	6.29	<0.001
HDL (mg/dL)	36.81	37.36	37.48	37.82	1.01	<0.001
BMI (kg/m ²)	28.96	28.85	28.80	28.71	0.25	<0.001
Waist Circumference (cm)	97.70	97.41	97.37	97.15	0.55	<0.001
Weight (kg)	80.49	80.37	79.97	79.72	0.77	<0.001

Table 7 represents mean changes across all study groups. Both of the intervention groups have shown great improvements. Improvements were greater than those of their respective control groups ($p < 0.001$). The improvement in glycemic control, liver enzymes, lipid profile, and anthropometric measures in the MAFLD group were comparable to the improvements in similar parameters of non-MAFLD intervention groups. On the other hand, the control groups did not show any major changes.

Table 7: Comparison of Mean Changes in Outcome Measures Across the Four Study Groups (Mean $\pm$ SD)

Variable	MAFLD + Intervention	MAFLD Control	No MAFLD + Intervention	No MAFLD + Control	Test	p-value
SBP (mmHg)	9.64 $\pm$ 3.36	2.59 $\pm$ 3.15	9.73 $\pm$ 3.04	2.96 $\pm$ 2.50	Kruskal-Wallis	<0.001
DBP (mmHg)	4.08 $\pm$ 1.92	0.87 $\pm$ 1.83	3.75 $\pm$ 2.15	1.25 $\pm$ 1.95	Kruskal-Wallis	<0.001
FBS (mg/dL)	25.20 $\pm$ 3.42	5.34 $\pm$ 3.68	24.59 $\pm$ 3.83	5.11 $\pm$ 3.84	One-way ANOVA	<0.001

Variable	MAFLD + Intervention	MAFLD Control	No MAFLD + Intervention	No MAFLD + Control	Test	p-value
PPBS (mg/dL)	34.35 ± 5.06	8.40 ± 4.96	35.00 ± 5.13	8.58 ± 5.35	Kruskal-Wallis	<0.001
HbA1c (%)	0.91 ± 0.14	0.20 ± 0.14	0.90 ± 0.14	0.20 ± 0.13	One-way ANOVA	<0.001
ALT (U/L)	21.80 ± 2.59	2.37 ± 2.88	17.50 ± 2.27	2.52 ± 3.16	Kruskal-Wallis	<0.001
AST (U/L)	17.55 ± 2.04	2.84 ± 2.64	15.04 ± 2.23	2.74 ± 2.40	One-way ANOVA	<0.001
ALP (U/L)	13.91 ± 1.72	1.88 ± 1.53	11.94 ± 1.97	1.77 ± 1.81	One-way ANOVA	<0.001
Triglycerides (mg/dL)	35.69 ± 4.34	6.72 ± 4.10	29.64 ± 4.42	6.29 ± 5.00	One-way ANOVA	<0.001
HDL (mg/dL)	4.18 ± 0.65	0.99 ± 0.64	4.01 ± 0.59	1.01 ± 0.68	One-way ANOVA	<0.001
BMI (kg/m ²)	1.17 ± 0.18	0.27 ± 0.20	1.18 ± 0.17	0.25 ± 0.18	One-way ANOVA	<0.001
Waist Circumference (cm)	3.09 ± 0.48	0.66 ± 0.41	2.97 ± 0.52	0.55 ± 0.49	One-way ANOVA	<0.001
Weight (kg)	3.44 ± 0.43	0.69 ± 0.48	3.58 ± 0.52	0.77 ± 0.47	One-way ANOVA	<0.001

Table 8 represents the overall effect of nutrition and lifestyle in the intervention group as compared to the control groups. Significant improvement in the BMI, body weight, glycemic parameters, liver enzymes, triglycerides, and HDL levels were observed for the intervention group.

Table 8: Effect of Nutrition and Lifestyle Intervention on Changes in Clinical Parameters Over 6 Months (Intervention vs. Control)

Outcome	Intervention (mean ± SD)	Control (mean ± SD)	Mean Difference	95% CI	P-value	Cohen's d
BMI change (kg/m ²)	-1.18 ± 0.18	-0.26 ± 0.19	-0.92	-0.96 to -0.88	<0.001	-5.05
Weight change (kg)	-3.51 ± 0.48	-0.73 ± 0.47	-2.78	-2.88 to -2.67	<0.001	-5.80
Waist circumference change (cm)	-3.03 ± 0.50	-0.61 ± 0.46	-2.42	-2.53 to -2.31	<0.001	-5.05
FBS change (mg/dL)	-24.89 ± 3.63	-5.22 ± 3.75	-19.67	-20.51 to -18.83	<0.001	-5.33
PPBS change (mg/dL)	-34.67 ± 5.09	-8.49 ± 5.14	-26.18	-27.34 to -25.02	<0.001	-5.12
HbA1c change (%)	-0.90 ± 0.14	-0.20 ± 0.13	-0.70	-0.73 to -0.67	<0.001	-5.19
ALT change (U/L)	-19.65 ± 3.25	-2.44 ± 3.01	-17.20	-17.91 to -16.49	<0.001	-5.49
AST change (U/L)	-16.29 ± 2.47	-2.79 ± 2.51	-13.50	-14.07 to -12.94	<0.001	-5.42
ALP change (U/L)	-12.92 ± 2.09	-1.83 ± 1.67	-11.10	-11.53 to -10.67	<0.001	-5.86

Outcome	Intervention (mean ± SD)	Control (mean ± SD)	Mean Difference	95% CI	P- value	Cohen's d
Triglycerides change (mg/dL)	-32.67 ± 5.32	-6.50 ± 4.56	-26.16	-27.29 to - 25.04	<0.001	-5.28
HDL change (mg/dL)	+4.09 ± 0.62	+1.00 ± 0.66	+3.10	+2.95 to +3.24	<0.001	+4.83

DISCUSSION

T2DM and MAFLD are closely interconnected and share common pathophysiology. But there is evidence of improvement in their existences if managed through lifestyle changes. Therefore, it is highly important to evaluate the effectiveness of comprehensive non-pharmacological interventions when it comes to management of these conditions. The present study proves the significance of a six-month structured nutrition and lifestyle intervention across multiple domains in T2DM patients. It has produced clinically meaningful and statistically significant results, irrespective of MAFLD status.

The intervention groups demonstrated substantially greater improvements than the control groups across most outcome measures. Large effect sizes were observed for several parameters, indicating a strong association between structured lifestyle intervention and metabolic improvement. Marked reduction in glycemic parameters was observed in the intervention groups. It has been established earlier that a high-protein diet and low glycemic index can significantly improve metabolic parameters in MAFLD patients. [6]

Similarly, studies have proven that change in lifestyle can directly improve liver enzyme levels, metabolic status, and anthropometric outcomes in patients with MAFLD. [7]

A reduction of 0.90-0.91% in HbA1c was observed in both intervention groups. These findings are in accordance with the study by Franz MJ et al., who reported significant reductions in body weight and glycemic parameters in overweight and obese adults with T2DM following structured lifestyle interventions. [8]

Noticeable reduction in level of liver enzymes in the MAFLD intervention group was achieved. It suggests a decrease in hepatic inflammation as well as improvement in hepatocellular integrity. The findings suggest a partial reversal of early hepatic injury.

Most importantly, there was a reduction in liver enzymes in the non-MAFLD intervention group. This indicates the metabolic benefits of dietary and lifestyle changes, which can also be detected even before any structural liver disease starts to develop. This concept aligns with the fact that metabolic intervention may prevent disease progression. [9] The observation aligns with the clinical practice recommendations proposed by Eslam M et al., which emphasize the importance of early metabolic intervention in preventing disease progression in MAFLD. [10] A reduction in triglycerides and an increase in HDL indicate improvement in the lipid profile. This further improves lipid metabolism and reduces cardiac risk to the patients. Enhanced insulin sensitivity, reduced hepatic de novo lipogenesis, and reverse cholesterol transport are mediated through the change in the approach towards management of the condition. [11]

A minimum change of lipid profile was observed in the control groups. This reinforces that a passive follow-up or standard care alone is insufficient in achieving metabolic improvements.

A major noticeable finding of the current study is the similar degree of improvements which were seen in MAFLD and non-MAFLD parameters. So, the benefits of structured lifestyle modifications are not limited to the patients with liver conditions only. It has a preventive potential in T2DM patients before hepatic involvement. Studies in the past have reported reductions in body weight and glycemic variables with lifestyle change programs in T2DM patients. [8] To sum up, studies have also shown a decrease in intrahepatic fat and improved metabolic values in patients with T2DM who were exposed to structured lifestyle modifications. [12]

Limitations

The limitations of the study can also be listed. Firstly, The study was conducted in a single urban centre, which limits its generalizability. Also, a period of six-months' intervention does not capture long-term sustainability of the outcomes. Although biochemical improvement was demonstrated, follow-up hepatic imaging was not performed. Self-assessment and self-reporting are subjected to recall and have chances of bias. Thus, future research should be focusing more on longer durations of interventions and should target more diverse geographic settings. They can even include hepatic imaging endpoints like controlled attenuation parameters and magnetic resonance imaging (MRI) based quantifications.

Conclusion

Clinically significant improvement can be achieved in patients with MAFLD and T2DM patients who are subjected to structured nutrition and lifestyle modifications. Robust improvements in metabolic, glycemic, hepatic, and lipid profiles can be obtained in patients with or without MAFLD. The effect of intervention is uniform across subgroups and can be substantially increased with standard care. All the findings strongly support the need for dietary and lifestyle modifications as a non-pharmacological strategy in the management of patients with MAFLD and T2DM.

Financial Support

Nil.

Conflicts of Interest

No conflicts of interest.

List of Abbreviations

MAFLD	Metabolic Dysfunction-Associated Fatty Liver Disease
T2DM	Type 2 Diabetes Mellitus
FBS	Fasting Blood Sugar
PPBS	Postprandial Blood Sugar
HbA1c	Glycated Hemoglobin
ALT	Alanine Aminotransferase
AST	Aspartate Aminotransferase
HDL	High-Density Lipoprotein
BMI	Body Mass Index
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
ALP	Alkaline Phosphatase
NABL	National Accreditation Board for Testing and Calibration Laboratories
SPSS	Statistical Package for the Social Sciences
ANOVA	Analysis of Variance
SD	Standard Deviation
CI	Confidence Interval
NAFLD	Non-Alcoholic Fatty Liver Disease
NASH	Non-Alcoholic Steatohepatitis
NF-κB	Nuclear Factor Kappa B
PPARγ	Peroxisome Proliferator-Activated Receptor Gamma
MRI	Magnetic Resonance Imaging
U/L	Units per Liter

REFERENCES

1. Eslam M, Newsome PN, Sarin SK, Anstee QM, Targher G, Romero-Gomez M, et al. A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. *J Hepatol* 2020;73:202-9.
2. Wainwright P, Byrne CD. Bidirectional relationships and disconnects between NAFLD and features of the metabolic syndrome. *Int J Mol Sci* 2016;17:367.
3. Tang A, Rabasa-Lhoret R, Castel H, Wartelle-Bladou C, Gilbert G, Massicotte-Tisluck K, et al. Effects of insulin glargine and liraglutide therapy on liver lipid content and body fat distribution in patients with type 2 diabetes. *Diabetes Care* 2023;38:1482-90.
4. Younossi ZM, Golabi P, de Avila L, Paik JM, Srishord M, Fukui N, et al. The global epidemiology of NAFLD and NASH in patients with type 2 diabetes: A systematic review and meta-analysis. *J Hepatol* 2019;71:793-801.
5. Lim JS, Mietus-Snyder M, Valente A, Schwarz JM, Lustig RH. The role of fructose in the pathogenesis of NAFLD and the metabolic syndrome. *Nat Rev Gastroenterol Hepatol* 2021;7:251-64.
6. Sun X, Feng R, Li Y, Lin S, Zhang W, Li Y, et al. Histidine supplementation alleviates inflammation in the adipose tissue of high-fat diet-induced obese rats via the NF-κB-and PPARγ-involved pathways. *Br J Nutr* 2022;112:477-85.
7. Chai W, Morin-Papunen L, Piltonen TT, Kortekaas JC, Mancini GF, Slotboom J, et al. Dietary interventions in MAFLD: A systematic review and meta-analysis. *J Hepatol* 2023;79:1170-84.
8. Franz MJ, Boucher JL, Rutten-Ramos S, VanWormer JJ. Lifestyle weight-loss intervention outcomes in overweight and obese adults with type 2 diabetes: A systematic review and meta-analysis of randomized clinical trials. *J Acad Nutr Diet* 2015;115:1447-63.
9. Eslam M, Sarin SK, Wong VW, Fan JG, Kawaguchi T, Ahn SH, et al. The Asian Pacific Association for the Study of the Liver clinical practice guidelines for the diagnosis and management of metabolic associated fatty liver disease. *Hepatol Int* 2020;14:889-919.
10. Mascaró CM, Bouzas C, Tur JA. Association between non-alcoholic fatty liver disease and Mediterranean lifestyle: A systematic review. *Nutrients* 2022;14:1mediterranean.
11. Jurek J, Szulinska M, Kregielska-Narozna M, Bogdanski P. Dietary interventions in MASLD—A narrative review. *Nutrients* 2025;17:234.
12. Abdelbasset WK, Tantawy SA, Kamel DM, Alqahtani BA, Soliman GS. A randomized controlled trial on the effectiveness of 8-week high-intensity interval exercise on intrahepatic triglycerides, visceral lipids, and health-related quality of life in diabetic obese patients with nonalcoholic fatty liver disease. *Medicine* 2019;98:e15638.