

## Effect of structured yoga practice on glycemic and hepatic outcomes in patients with Co-existing Type 2 Diabetes Mellites and Non -Alcoholic Fatty Liver Disease

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### ABSTRACT

**Background:** Non-alcoholic fatty liver disease and type 2 diabetes mellitus often coexist and have similar metabolic risk factors. A key component of management is still changing one's lifestyle. Hepatic and metabolic parameters may be improved by yoga, as an adjuvant therapy to the standard. **Aims and objective:**The current study aim was to evaluate the effect of yoga asanas on biochemical parameters of diabetes (FBS, PPBS, HbA1c) and NAFLD (ALT, AST). **Methodology:** This study was designed as a quasi-experiment and conducted at ESIC Medical College and Hospital, Faridabad. Total 160 participants with T2DM and NAFLD were recruited after obtaining the inform consent and ethics approval. The enrolled participant further divided in the two -group intervention and control group. Intervention group included the addition of the designed yogic program to the standard care of treatment and control group included the participant with standard care of treatment alone. The enrolled participants are followed up for 12 months. Biochemical parameter (Fasting Blood Sugar, Post-prandial Blood Sugar and HbA1c as glycemic and ALT and AST as Hepatic) is evaluated at month 3, month 6, month 9 and month 12. **Result:** The Intervention group showed significant reduction in FBS ( $153.18 \pm 46.34$  to  $96.32 \pm 21.21$ ), PPBS ( $261.85 \pm 56.23$  to  $162.10 \pm 38.42$  mg/dL) and HbA1c ( $8.19 \pm 0.76\%$  to  $6.52 \pm 0.66\%$ ) at 12 months ( $p < 0.0001$ ), with significant inter-group differences for HbA1c at all follow-ups. ALT decreased from  $62.58 \pm 19.33$  to  $58.47 \pm 15.37$  U/L with significant inter-group differences at Months 9 and 12, while AST showed comparable changes between groups ( $p > 0.05$ ). Overall, the intervention was associated with significant improvements in glycemic parameters and ALT levels over 12 months. **Conclusion:** Over 12 months, period of follow up, the intervention group showed good decrease in liver enzymes and significant long-lasting improvements in glycemic parameters (FBS, PPBG, and HbA1c). These results indicate that individuals with coexisting T2DM and NAFLD can benefit from an integrated approach that effectively improves both metabolic and hepatic outcomes.

### KEYWORDS

Yoga, Type 2 Diabetes Mellitus(T2DM), Non-Alcoholic Fatty Liver Disease (NAFLD), HbA1c, ALT, AST, Glycemic Control, Hepatic Parameter

### INTRODUCTION

T2DM is a long-term metabolic condition marked by beta-cell dysfunction and persistence hyperglycemia brought on by insulin resistance (1).

The requirement for adjunct lifestyle interventions is highlighted by the facts that optimum long term glycemic management is remain difficult to achieve despite pharmaceutical advancements.

Literature supports that yoga-based therapies have been shown to enhance anthropometric measures, cardiovascular risk markers, insulin resistance indices, lipid profile parameters (2), inflammatory mediators and fasting blood glucose (3, 4, and 5).

The hepatic manifestation of metabolic dysfunction known as NAFLD is characterized by an

The hepatic manifestation of metabolic dysfunction known as Non-Alcoholic Fatty Liver Disease (NAFLD) is characterized by an redundant of fat buildup in hepatocyte which do not consume a hepatocytes that do not consume significant amount of alcohol. Further, T2DM and obesity, its prevalence is rising (6,7).

Modification in the lifestyle is still crucial for therapy, regular structured exercise helps in improvement of metabolic profiles, hepatic steatosis, and liver enzymes (8,9,10,11).

The link between T2DM and NAFLD is reciprocal: Diabetes speeds up the development of steatosis whereas NAFLD

exacerbates insulin resistance and glycemic control (12,13).

Chronic inflammation, oxidative stress, sympathetic hyperactivity and adipose dysfunction are very known pathways. Yoga has been found to improve inflammatory markers like IL-6 (14,15), heart rate variability, psychological well-being (16), and cardiometabolic health (17,18) by combining asanas, relaxation and pranayama,

There is still very rare long-term data availability on patients who have both T2DM and NAFLD. Hence this research assesses how organized yoga works on hepatic (ALT, AST) and Glycemic (FBS, PPBS and HbA1c) parameters.

#### **Aim & Objective(s)**

To evaluate the effect of yoga asanas on biochemical parameters of diabetes (FBS, PPBS, HbA1c) and NAFLD (ALT, AST).

### **MATERIAL & METHODS**

#### **Study type & study Design**

This study was conducted as a prospective quasi experiment study.

#### **Study Setting**

The study was conducted at the clinical setting of ESIC Medical College and Hospital, Faridabad.

#### **Study population**

A total of 160 participants were enrolled in the study and were allocated in a 1:1 ratio into two groups, comprising 80 participants in the intervention group and 80 participants in the control group.

#### **Study Duration**

Participants were recruited between April 10, 2024, and Dec 14, 2024, with follow-up and post-intervention assessments completed by Dec 2025.

#### **Sample Size calculation**

The sample size was calculated using a two-sided confidence level of 95% and a power of 80%, with an equal allocation ratio (1:1) between intervention and control groups. Based on an expected outcome proportion of 20% in the control group and 56% in the intervention group, the minimum required sample size was estimated to be 66 participants (33 per group) using the Fleiss method with continuity correction. After accounting for an anticipated dropout rate of 20%, the required sample size was increased to 40 participants per group. However, to enhance the statistical power and generalizability of the findings, a total of 160 participants were enrolled in the study, with 80 participants allocated to each group.

#### **Inclusion and exclusion criteria**

Eligible participants were men and women aged 30 to 60 years (both inclusive) who had been diagnosed with Type 2 Diabetes Mellitus (T2DM) and Non-Alcoholic Fatty Liver Disease (NAFLD) based on their medical history, physical examination, and relevant laboratory investigations. Participant with FibroScan grading between F0 and F2 were included. Glycated Hemoglobin (HbA1c) levels greater than 7% and up to 10% at baseline were considered as one of the inclusion criteria. All enrolled participants were capable of understanding and complying with the study-related instructions and prohibitions as outlined in the protocol. Participants were

excluded if they had Type 1 diabetes mellitus, advanced liver disease (FibroScan grade  $\geq$  F3), liver cirrhosis, viral hepatitis, alcoholic liver disease, or other chronic liver disorders. Persons with significant cardiovascular, renal, respiratory, or psychiatric illnesses that could interfere with study participation were also excluded. Pregnant or Lactating women, person with recent major surgical history, or participating in another clinical study.

#### **Data Collection Method**

Data were collected using a structured pre-designed form after obtaining written informed consent from all participant. Baseline assessment includes demographic details, medical history, physical examination, vital signs, and biochemical investigation (FBS, PPBS, HbA1c, ALT, and AST). Eligible participants aged between 30-60 years with diagnosed T2DM and NAFLD were recruited in two groups Intervention (received structured yoga asanas (45–60 minutes/session) for 12 months in addition to standard care) and control (received standard medical management alone). Follow-up assessment were conducted at 3, 6, 9, and 12 months to record anthropometric measures and biochemical parameters. Final clinical evaluation was performed at 12 months to assess overall treatment outcomes compared with baseline.

#### **Ethical and Inform Consent**

Ethical approval was obtained from the institutional ethics committee of ESIC Medical College, Faridabad (17 October 2023; Approval No. 134 X/11/13/2023-IEC/DHR/99). The study was also prospectively registered at Indian Clinical Trials Registry (CTRI/2024/03/064485; 20 March 2024). Written informed consent was obtained from all participants prior to enrolment.

#### **Data Analysis and Software**

Data were recorded into Microsoft Excel and analyzed using SPSS version 30. Categorical variable are presented as frequency and percentages while Continuous variable were expressed as mean  $\pm$  standard deviation and compared using paired/independent t-tests (or non-parametric equivalents where appropriate). p-value  $<0.05$  was considered statistically significant.

#### **Biochemical Parameter assessment**

Venous blood samples were collected for the testing of fasting blood sugar at baseline, 3, 6, 9 and 12 months. Post-prandial samples were obtained 2 hours after a standard meal. Testing were done using the glucose oxidase–peroxidase (GOD-POD) method for FBS. standardized high-performance liquid chromatography (HPLC) were used to analyse the Glycated hemoglobin (HbA1c). Alanine transaminase (ALT) and aspartate transaminase (AST) were analyzed using automated biochemistry analyzers. All investigations were performed in a NABL-accredited central laboratory following standard operating procedures and quality control protocols.

### **RESULTS**

**Demographic Characteristics and Disease History:** Out of 160 enrolled participants, 150 participants completed the study and were included in the final analysis, with 74 participants in the Intervention and 76 in the control group.

With respect to gender distribution, females constituted a slightly higher proportion in both groups, accounting for 55.41% in the Intervention and 60.53% in the control group. Males represented 44.59% and 39.47% in the yoga and non-yoga groups, respectively, indicating comparable gender distribution at baseline.

The mean age of participants was similar in both groups ( $47.03 \pm 8.42$  years in the yoga group and  $47.49 \pm 8.06$  years in the non-yoga group), suggesting that the study population predominantly consisted of middle-aged individuals and that the two groups were well matched. The mean height was  $165.59 \pm 9.53$  cm in the yoga group and  $163.96 \pm 8.63$  cm in the non-yoga group, showing no significant baseline difference.

More than half of the participants in the yoga group (52.70%) and 40.79% in the non-yoga group reported a family history of diabetes, indicating a substantial genetic predisposition in both groups. None of the participants reported alcohol consumption, consistent with the diagnosis of non-alcoholic fatty liver disease (NAFLD). Physical examination findings were normal in all participants, and none had previously engaged in structured lifestyle modification programs prior to enrolment.

Regarding the duration of Type 2 Diabetes Mellitus, approximately one-third of participants in both groups had a disease duration of less than one year (32.43% in yoga vs. 34.21% in non-yoga), around 27–30% had diabetes for 1–4 years, and nearly 38–39% had a duration of  $\geq 5$  years, demonstrating comparable disease chronicity between groups.

All participants were confirmed cases of NAFLD based on Fibroscan assessment at baseline, with grading ranging from 0 to 2 as per study inclusion criteria. Participants with other liver diseases were excluded, ensuring a homogenous study population with early-stage NAFLD associated with Type 2 Diabetes Mellitus.

Overall, the demographic characteristics and disease history were well balanced between the two groups, thereby reducing potential baseline confounding and strengthening the internal validity of the study findings. Assessment of Biochemical Parameter of type 2 diabetes **Glycemic Parameters (FBS, PPBS, and HbA1c)**

The mean fasting blood sugar levels in the intervention group decreased progressively from  $153.18 \pm 46.34$  mg/dL to  $129.27 \pm 50.04$  mg/dL at month 3,  $121.58 \pm 30.64$  mg/dL at Month 6,  $92.14 \pm 30.22$  mg/dL at month 9 and  $96.32 \pm 21.21$  mg/dL at Month 12. These reductions were statistically significant within the group at all follow-up periods ( $p < 0.0001$ ). In contrast, the control group showed FBS levels of  $186.33 \pm 52.62$  mg/dL at baseline,  $189.82 \pm 61.38$  mg/dL at Month 3 ( $p = 0.23257$ ),  $166.82 \pm 58.96$  mg/dL at Month 6 ( $p < 0.0001$ ),  $143.96 \pm 51.27$  mg/dL at Month 9 ( $p < 0.0001$ ), and  $132.51 \pm 40.89$  mg/dL at Month 12 ( $p < 0.0001$ ). Inter-group comparison showed significant differences at Month 3 ( $p < 0.0001$ ), Month 6 ( $p = 0.0257783$ ), and Month 9 ( $p = 0.0005275$ ), whereas no significant difference was observed at Month 12 ( $p = 0.9310949$ ).

Similarly, post-prandial blood sugar (PPBS) levels in the intervention group decreased significantly from  $261.85 \pm 56.23$  mg/dL at baseline to  $246.55 \pm 59.27$  mg/dL at Month 3,  $188.20 \pm 39.88$  mg/dL at Month 6,  $158.64 \pm$

$39.90$  mg/dL at Month 9, and  $162.10 \pm 38.42$  mg/dL at Month 12 ( $p < 0.0001$  at all follow-ups). The control group recorded PPBS values of  $258.37 \pm 69.59$  mg/dL at baseline,  $265.22 \pm 70.27$  mg/dL at Month 3 ( $p = 0.04573$ ),  $239.12 \pm 75.27$  mg/dL at Month 6 ( $p < 0.0001$ ),  $213.07 \pm 75.45$  mg/dL at Month 9 ( $p < 0.0001$ ), and  $195.90 \pm 60.19$  mg/dL at Month 12 ( $p < 0.0001$ ). Inter-group comparison revealed statistically significant differences at Month 3 ( $p = 0.0000079$ ), Month 6 ( $p < 0.0001$ ), Month 9 ( $p < 0.0001$ ), and Month 12 ( $p = 0.0000047$ ), indicating greater improvement in the intervention group compared to the control group.

The mean Glycated Hemoglobin (HbA1c %) levels in the intervention group decreased progressively from  $8.19 \pm 0.76\%$  at baseline to  $8.04 \pm 0.77\%$  at Month 3,  $7.81 \pm 0.76\%$  at Month 6,  $6.96 \pm 0.67\%$  at Month 9, and  $6.52 \pm 0.66\%$  at Month 12. These reductions were statistically significant within the intervention group at all follow-up periods ( $p < 0.0001$ ). In contrast, the control group showed minimal change from  $8.25 \pm 0.84\%$  at baseline to  $8.26 \pm 0.85\%$  at Month 3 ( $p = 0.43283$ ) and  $8.26 \pm 0.86\%$  at Month 6 ( $p = 0.37777$ ), but demonstrated significant reductions at Month 9 ( $7.76 \pm 0.54\%$ ;  $p < 0.0001$ ) and Month 12 ( $7.51 \pm 0.57\%$ ;  $p < 0.0001$ ). Inter-group comparison revealed statistically significant differences at Months 3, 6, 9, and 12 ( $p < 0.0001$ ), indicating greater improvement in HbA1c levels in the intervention group compared to the control group.

#### **Assessment of Biochemical Parameter of Non-Alcoholic Fatty Liver Disease**

Regarding Alanine Aminotransferase (ALT), the intervention group had baseline levels of  $62.58 \pm 19.33$  U/L, which increased to  $69.38 \pm 21.67$  U/L at Month 3 and  $69.45 \pm 22.06$  U/L at Month 6 ( $p < 0.0001$ ), then decreased to  $61.68 \pm 20.21$  U/L at Month 9 ( $p = 0.5348$ ) and  $58.47 \pm 15.37$  U/L at Month 12 ( $p = 0.0074$ ). The control group showed baseline levels of  $43.90 \pm 14.40$  U/L, increasing to  $51.01 \pm 19.96$  U/L at Month 3 and  $51.12 \pm 20.32$  U/L at Month 6 ( $p < 0.0001$ ), followed by reductions to  $48.50 \pm 20.20$  U/L at Month 9 ( $p = 0.00137$ ) and  $48.22 \pm 16.30$  U/L at Month 12 ( $p = 0.00072$ ). Inter-group comparison was not significant at Months 3 ( $p = 0.9460240$ ) and 6 ( $p = 0.9475256$ ), but became significant at Month 9 ( $p = 0.0117342$ ) and highly significant at Month 12 ( $p = 0.0000160$ ).

For Aspartate Aminotransferase (AST), the intervention group showed levels of  $40.66 \pm 16.15$  U/L at baseline, rising to  $47.91 \pm 17.68$  U/L at Month 3 and  $47.31 \pm 16.48$  U/L at Month 6 ( $p = 0.0000$ ), then decreasing to  $38.41 \pm 16.18$  U/L at Month 9 ( $p = 0.0326$ ) and  $35.04 \pm 12.59$  U/L at Month 12 ( $p = 0.0002$ ). The control group had baseline AST levels of  $33.62 \pm 9.50$  U/L, increasing to  $39.36 \pm 12.47$  U/L at Month 3 and  $39.46 \pm 12.90$  U/L at Month 6 ( $p = 0.00000$ ), and subsequently decreasing to  $30.33 \pm 12.63$  U/L at Month 9 ( $p = 0.00185$ ) and  $30.40 \pm 8.78$  U/L at Month 12 ( $p = 0.00052$ ). However, inter-group comparisons for AST were not statistically significant at Months 3 ( $p = 0.2726449$ ), 6 ( $p = 0.5042407$ ), 9 ( $p = 0.4677235$ ), and 12 ( $p = 0.3169715$ ), indicating comparable changes between the two groups over time.

In conclusion, Alanine Aminotransferase (ALT) levels initially increased in both groups up to Month 6, followed

by a reduction toward Month 12. At Months 9 (p = 0.0117342) and 12 (p = 0.0000160), significant inter-group differences were found, suggesting that the intervention group had improved over time. Both groups' aspartate aminotransferase (AST) levels exhibited a

similar pattern of initial spike and subsequent reduction. Comparable changes between the intervention and control groups were suggested by the fact that intergroup comparisons at all follow-up periods were not statistically significant (p > 0.05).

**Table 1. Baseline Demographic Characteristics of Participants in the Intervention and Control Groups**

Characteristic	Statistics	Group	
		Intervention (N=74)	Control (N=76)
<b>Gender</b>			
<b>Female</b>	n (%)	41/74 (55.41%)	46/76 (60.53%)
<b>Male</b>	n (%)	33/74 (44.59%)	30/76 (39.47%)
<b>Age (in Years)</b>	Mean ± SD	47.03 ± 8.42	47.49 ± 8.06

Values are presented as mean ± standard deviation (SD) for continuous variables and number (percentage) for

categorical variables. N = total number of participants in each group. SD = Standard deviation.

**Table 2. Comparison of Fasting Blood Sugar (FBS) and Post-Prandial Blood Sugar (PPBS) Levels Between Intervention and Control Groups Over 12 Months**

Time Point	FBS (mg/dL) Mean ± SD	Contr ol	p-value Intra group	Interventi on	Contr ol	p-value Inter Group	PPBS (mg/dL) Mean ± SD	Contr ol	p-value Intra group	Interventi on	Contr ol	p-value Inter Group
<b>Baseline</b>	153.18 ± 46.34	186.3 ± 52.62	-	-	-	-	261.85 ± 56.23	258.3 ± 69.59	-	-	-	-
<b>Month 3</b>	129.27 ± 50.04	189.8 ± 61.38	<0.0001	0.232	57	<0.0001	246.55 ± 59.27	265.2 ± 70.27	<0.0001	0.045	73	0.00000
<b>Month 6</b>	121.58 ± 30.64	166.8 ± 58.96	<0.0001	<0.00	01	0.02577	188.20 ± 39.88	239.1 ± 75.27	<0.0001	<0.00	01	<0.0001
<b>Month 9</b>	92.14 ± 30.22	143.9 ± 51.27	<0.0001	<0.00	01	0.00052	158.64 ± 39.90	213.0 ± 75.45	<0.0001	<0.00	01	<0.0001
<b>Month 12</b>	96.32 ± 21.21	132.5 ± 40.89	<0.0001	<0.00	01	0.93109	162.10 ± 38.42	195.9 ± 60.19	<0.0001	<0.00	01	0.00000

Values are expressed as mean ± standard deviation (SD). FBS = Fasting Blood Sugar; PPBS = Post-Prandial Blood Sugar. Intra-group p-values represent changes from baseline within each group. Inter-group p-values

represent comparisons between intervention and control groups at each time point. A p-value < 0.05 was considered statistically significant.

**Table 3. Comparison of Alanine Aminotransferase (ALT) Levels Between Intervention and Control Groups Over 12 Months**

Time Point	Alanine Aminotransferase (ALT ± SD)		p-value Intra group		p-value Inter Group
	Intervention	Control	Intervention	Control	
<b>Baseline</b>	62.58 ± 19.33	43.90 ± 14.40	-	-	-
<b>Month 3</b>	69.38 ± 21.67	51.01 ± 19.96	<0.0001	<0.0001	0.9460240
<b>Month 6</b>	69.45 ± 22.06	51.12 ± 20.32	<0.0001	<0.0001	0.9475256
<b>Month 9</b>	61.68 ± 20.21	48.50 ± 20.20	0.5348	0.00137	0.0117342
<b>Month 12</b>	58.47 ± 15.37	48.22 ± 16.30	0.0074	0.00072	0.0000160

Values are presented as mean ± standard deviation (SD). ALT = Alanine Aminotransferase (U/L). Intra-group p-values indicate comparison with baseline values within the same group. Inter-group p-values indicate

comparison between intervention and control groups at corresponding time points. A p-value < 0.05 was considered statistically significant.

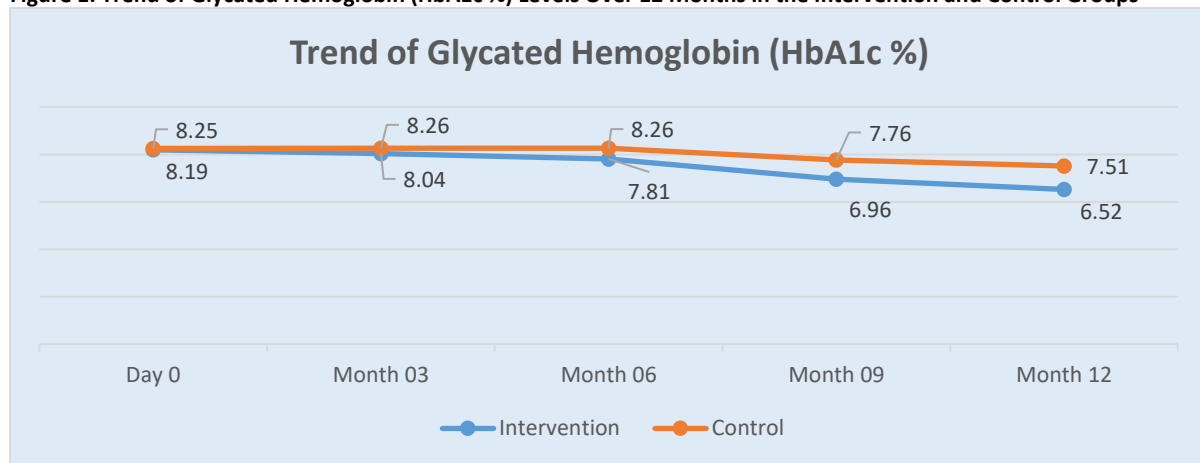
**Table 4. Comparison of Aspartate Aminotransferase (AST) Levels Between Intervention and Control Groups Over 12 Months**

Time Point	Aspartate Aminotransferase(AST ± SD)		p-value Intra group		p-value Inter Group
	Intervention	Control	Intervention	Control	
<b>Baseline</b>	40.66 ± 16.15	33.62 ± 9.50	-	-	-
<b>Month 3</b>	47.91 ± 17.68	39.36 ± 12.47	0.0000	0.00000	0.2726449
<b>Month 6</b>	47.31 ± 16.48	39.46 ± 12.90	0.0000	0.00000	0.5042407
<b>Month 9</b>	38.41 ± 16.18	30.33 ± 12.63	0.0326	0.00185	0.4677235
<b>Month 12</b>	35.04 ± 12.59	30.40 ± 8.78	0.0002	0.00052	0.3169715

Values are presented as mean ± standard deviation (SD). AST = Aspartate Aminotransferase (U/L). Intra-group p-values indicate changes from baseline within each group.

Inter-group p-values represent between-group comparisons at each time point. A p-value < 0.05 was considered statistically significant.

**Figure 1. Trend of Glycated Hemoglobin (HbA1c %) Levels Over 12 Months in the Intervention and Control Groups**



The graph illustrates the longitudinal changes in mean HbA1c (%) levels at baseline, 3, 6, 9, and 12 months in both intervention and control groups. A progressive reduction in HbA1c was observed in the intervention group over the study period, whereas comparatively lesser improvement was noted in the control group. Values represent mean ± standard deviation (SD). Statistical significance was considered at p < 0.05.

**DISCUSSION**

The current study supported the metabolic interrelationship between Type 2 Diabetes Mellitus (T2DM) and Non-Alcoholic Fatty Liver Disease (NAFLD) by showing a significant improvement in glycemc parameters (FBS, PPBS, and HbA1c) in the intervention group over a 12-month period, along with favorable changes in hepatic enzymes. Effective glycemc control is reflected in the progressive decrease in fasting blood sugar from 153.18 ± 46.34 mg/dL to 96.32 ± 21.21 mg/dL and post-prandial blood sugar from 261.85 ± 56.23 mg/dL to 162.10 ± 38.42 mg/dL. These findings from the UK Prospective Diabetes Study (UKPDS), which showed that better glycemc control lowers microvascular complications in T2DM [19,20].

The long-term advantages of consistent glucose control were also demonstrated by the Diabetes Control and Complications Trial (DCCT) and its follow-up trial, the Epidemiology of Diabetes Interventions and Complications (EDIC) [21,22].

Clinically relevant is the considerable drop in HbA1c from 8.19 ± 0.76% to 6.52 ± 0.66%. According to Stratton et al.,

there is a 21% decrease in diabetes-related outcomes for every 1% drop in HbA1c [20].

Individualized objectives are advised, although large trials like ACCORD, ADVANCE, and VADT further demonstrated the significance of strict glycemc control [23–25].

The higher improvement in post-prandial glucose is consistent with research by Monnier et al. [26], who highlighted the substantial contribution of post-prandial hyperglycemia to the total glycemc burden, especially in patients with moderately increased HbA1c. The International Diabetes Federation also highlights post-prandial glucose as a critical therapeutic target [27].

In terms of liver markers, levels of ALT and AST increases until Month 6 and then decreased by Month 12. According to the AASLD practice guidelines, elevated ALT is closely linked to insulin resistance and hepatic steatosis [28].

Hepatic inflammation may have improved, as evidenced by the notable intergroup difference in ALT between Months 9 and 12. Enhancing insulin sensitivity lowers liver enzyme levels in NAFLD patients, according to research by Marchesini et al. [29]. Histological and biochemical improvements in NAFLD with improved metabolic control were also described by Sanyal et al. [30].

It is commonly known that T2DM and NAFLD are pathophysiologically related. Elevated transaminases are a result of oxidative stress, inflammatory cytokine release, and hepatic fat formation, all of which are encouraged by insulin resistance [31,32]. According to Targher et al. and Bellentani et al., NAFLD is quite

common in people with type 2 diabetes and is strongly associated with inadequate glycemic management [31,33]. Musso et al. and Younossi et al. conducted meta-analyses and came to the additional conclusion that metabolic risk factor-targeting therapies greatly improve liver enzymes and the course of the disease in NAFLD [34,35].

Thus, the parallel improvement in glycemic indices and ALT levels observed in the present study supports the concept that comprehensive metabolic management favorably impacts both diabetic and hepatic biochemical parameters.

#### CONCLUSION

The intervention produced significant and sustained reductions in FBS, PPBS, and HbA1c over 12 months, with greater improvement compared to the control group. The reduction in HbA1c to near-target levels indicates clinically meaningful long-term glycemic control and potential reduction in diabetes-related complications. Additionally, liver enzyme levels, particularly ALT, showed significant improvement over time, suggesting beneficial effects on hepatic function. Overall, the findings reinforce the strong metabolic link between T2DM and NAFLD and highlight the importance of integrated therapeutic strategies targeting both glycemic control and hepatic health.

#### RECOMMENDATION

Further large-scale randomized studies are recommended to validate these findings and support the integration of yoga into evidence-based clinical guidelines.

#### LIMITATION OF THE STUDY

The quasi-experimental design may limit generalizability. Baseline differences in some biochemical parameters and reliance on participant-reported adherence may introduce potential bias. Future randomized controlled trials with larger, multi-centric samples would further strengthen the evidence base.

#### RELEVANCE OF THE STUDY

The coexistence of T2DM and NAFLD complicates metabolic management, often rendering standard therapies insufficient for long-term control. This study evaluates a 12-month structured yoga program as an adjunct to traditional care to address these challenges. Results indicate significant improvements in blood glucose levels, liver enzymes, and insulin sensitivity. As a holistic mind-body intervention, yoga provides a safe, cost-effective, and culturally acceptable strategy. Integrating yoga into clinical practice offers a sustainable approach to enhancing both metabolic and hepatic health in high-risk patients.

#### AUTHORS CONTRIBUTION

All authors have contributed equally.

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Nil

#### CONFLICT OF INTEREST

There are no conflicts of interest.

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#### DECLARATION OF GENERATIVE AI AND AI ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

The authors declare that only minimal assistance from generative AI tools was utilized during the preparation of this manuscript. Such assistance was limited to basic language editing and formatting support and did not influence the scientific content, study design, data analysis, interpretation of results, or conclusions. All scientific work, analysis, and final decisions were performed independently by the authors.

#### REFERENCES

1. Zhao X, An X, Yang C, Sun W, Ji H, Lian F. The crucial role and mechanism of insulin resistance in metabolic disease. *Front Endocrinol (Lausanne)*. 2023;14:1149239.
2. Chen S, Deng S, Liu Y, Yin T. Effects of yoga on blood glucose and lipid profile of type 2 diabetes patients without complications: a systematic review and meta-analysis. *Front Sports Act Living*. 2022;4:900815.
3. Gowri MM, Rajendran J, Srinivasan AR, Bhavanani AB, Meena R. Impact of an integrated yoga therapy protocol on insulin resistance and glycemic control in patients with type 2 diabetes mellitus. *Rambam Maimonides Med J*. 2022;13(1):e0005.
4. Sharma S, Bhardwaj S, Gupta A, Katoch VM, Sharma KK, Gupta R. Influence of 24-week yoga intervention on cardiovascular risk factors and inflammatory markers in type 2 diabetes. *Int J Yoga*. 2023;16(1):27–33.
5. Kaur N, Majumdar V, Nagarathna R, Malik N, Anand A, Nagendra HR. Diabetic yoga protocol improves glycemic, anthropometric and lipid levels in high-risk individuals for diabetes: a randomized controlled trial from Northern India. *Diabetol Metab Syndr*. 2021;13:1–10.
6. Tabaeian SP, Rezapour A, Azari S, et al. Prevalence of non-alcoholic fatty liver disease in Iran: a systematic review and meta-analysis. *J Clin Exp Hepatol*. 2024;14(1):101209.
7. Rong L, Zou J, Ran W, Qi X, Chen Y, Cui H, et al. Advancements in the treatment of non-alcoholic fatty liver disease (NAFLD). *Front Endocrinol (Lausanne)*. 2023;13:1087260.
8. Stine JG, Long MT, Corey KE, et al. Physical activity and nonalcoholic fatty liver disease: a roundtable statement from the American College of Sports Medicine. *Med Sci Sports Exerc*. 2023;55(9):1717–.
9. Hallsworth K, Adams LA. Lifestyle modification in NAFLD/NASH: facts and figures. *JHEP Rep*. 2019;1(6):468–79.
10. Huang M, Yang J, Wang Y, Wu J. Comparative efficacy of different exercise modalities on metabolic profiles and liver

- functions in non-alcoholic fatty liver disease: a network meta-analysis. *Front Physiol.* 2024;15:1428723.
11. Nath P, Panigrahi MK, Sahu MK, et al. Effect of exercise on NAFLD and its risk factors: comparison of moderate versus low-intensity exercise. *J Clin Transl Hepatol.* 2020;8(2):120.
  12. Kumar V, Xin X, Ma J, Tan C, Osna N, Mahato RI. Therapeutic targets, novel drugs, and delivery systems for diabetes-associated NAFLD and liver fibrosis. *Adv Drug Deliv Rev.* 2021;176:113888.
  13. Cheng PN, Chen WJ, Hou CJ, Lin CL, Chang ML, Wang CC, Chang WT, Wang CY, Lin CY, Hung CL, Peng CY, Yu ML, Chao TH, Huang JF, Huang YH, Chen CY, Chiang CE, Lin HC, Li YH, Lin TH, Kao JH, Wang TD, Liu PY, Wu YW, Liu CJ. Taiwan Association for the Study of the Liver–Taiwan Society of Cardiology Taiwan position statement for the management of metabolic dysfunction-associated fatty liver disease and cardiovascular diseases. *Clin Mol Hepatol.* 2024;30(1):16-36. doi:10.3350/cmh.2023.0315. PMID: 37793641; PMCID: PMC10776290.
  14. Tyagi A, Cohen M. Yoga and heart rate variability: a comprehensive review of the literature. *Int J Yoga.* 2016;9(2):97–113.
  15. Nugent NR, Brick L, Armev MF, Tyrka AR, Ridout KK, Uebelacker LA. Benefits of yoga on IL-6: findings from a randomized controlled trial of yoga for depression. *Behav Med.* 2021;47(1):21–30.
  16. Park CL, Finkelstein-Fox L, Groessl EJ, Elwy AR, Lee SY. Exploring how different types of yoga change psychological resources and emotional well-being across a single session. *Complement Ther Med.* 2020;49:102354.
  17. Hossein-Nezhad A, Yarjoo B, Niketeghad G, et al. Cyclic yoga for the prevention of age-related sarcopenia. *Springer.* 2016:S283–S283.
  18. Momeni S, Mahdioun SS, Aghayari A. Cyclic yoga improves anthropometric indices, musculoskeletal disorders, and blood pressure in middle-aged women. *J Bodyw Mov Ther.* 2025;41:152–8.
  19. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes. *Lancet.* 1998;352(9131):837–53.
  20. Stratton IM, Adler AI, Neil HAW, Matthews DR, Manley SE, Cull CA, et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35). *BMJ.* 2000;321(7258):405–12.
  21. Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med.* 1993;329:977–86.
  22. Nathan DM, Cleary PA, Backlund JYC, Genuth SM, Lachin JM, Orchard TJ, et al. Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. *N Engl J Med.* 2005;353:2643–53.
  23. ACCORD Study Group. Effects of intensive glucose lowering in type 2 diabetes. *N Engl J Med.* 2008;358:2545–59.
  24. ADVANCE Collaborative Group. Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. *N Engl J Med.* 2008;358:2560–72.
  25. Duckworth W, Abraira C, Moritz T, Reda D, Emanuele N, Reaven PD, et al. Glucose control and vascular complications in veterans with type 2 diabetes. *N Engl J Med.* 2009;360:129–39.
  26. Monnier L, Lapinski H, Colette C. Contributions of fasting and postprandial plasma glucose increments to overall hyperglycemia. *Diabetes Care.* 2003;26(3):881–5.
  27. International Diabetes Federation. Guideline for management of postmeal glucose. Brussels: IDF; 2011.
  28. Chalasani N, Younossi Z, Lavine JE, Diehl AM, Brunt EM, Cusi K, et al. The diagnosis and management of non-alcoholic fatty liver disease: Practice guideline by AASLD. *Hepatology.* 2012;55(6):2005–23.
  29. Marchesini G, Brizi M, Bianchi G, Tomassetti S, Bugianesi E, Lenzi M, et al. Nonalcoholic fatty liver disease: A feature of the metabolic syndrome. *Diabetes.* 2001;50(8):1844–50.
  30. Sanyal AJ, Chalasani N, Kowdley KV, McCullough A, Diehl AM, Bass NM, et al. Pioglitazone, vitamin E, or placebo for nonalcoholic steatohepatitis. *N Engl J Med.* 2010;362:1675–85.
  31. Targher G, Bertolini L, Padovani R, Rodella S, Tessari R, Zenari L, et al. Prevalence of NAFLD and its association with cardiovascular disease in type 2 diabetes. *Diabetes Care.* 2007;30(5):1212–8.
  32. Bugianesi E, McCullough AJ, Marchesini G. Insulin resistance: A metabolic pathway to chronic liver disease. *Hepatology.* 2005;42(5):987–1000.
  33. Bellentani S, Scaglioni F, Marino M, Bedogni G. Epidemiology of NAFLD. *Dig Dis.* 2010;28(1):155–61.
  34. Musso G, Gambino R, Cassader M, Pagano G. Meta-analysis: Natural history of NAFLD and diagnostic accuracy of non-invasive tests. *Ann Med.* 2011;43(8):617–49.
  35. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of NAFLD. *Hepatology.* 2016;64(1):73–84.