

Influence of 24-Week Yoga Intervention on Cardiovascular Risk Factors and Inflammatory Markers in Type 2 Diabetes

Abstract

Background and Aims: Persistent hyperglycemia, dyslipidemia, inflammation, and oxidative stress are important in cardiovascular risk in type-2 diabetes mellitus (DM). To evaluate the effect of 24-week yoga intervention on anthropometry and biochemical markers in DM patients, we performed a study. **Methods:** A hospital-based prospective randomized study in 104 participants with DM divided into control ($n = 52$) and intervention ($n = 52$) groups was performed. Patients in the intervention group performed 40 min of multifaceted individualized yoga exercises 5 days/week for 24 weeks. Anthropometric measurements and biochemical analysis were performed at baseline and after 24 weeks in both groups. Descriptive statistics are reported. **Results:** Baseline characteristics were similar in both groups. At 24 weeks, participants in the intervention versus controls had lower body mass index (25.6 ± 2.9 vs. 28.0 ± 3.2 kg/m²), waist-hip ratio (0.94 ± 0.06 vs. 0.99 ± 0.05), systolic blood pressure (121.2 ± 11.7 vs. 139.3 ± 19.1 mmHg), fasting glucose (142.7 ± 45.3 vs. 175.7 ± 45.4 mg/dL), glycated hemoglobin (7.2 ± 1.8 vs. $9.4 \pm 1.9\%$), low-density lipoprotein cholesterol (167.5 ± 38.1 vs. 192.2 ± 51.4 mg/dL), nonhigh-density lipoprotein cholesterol (136.8 ± 35.3 vs. 158.6 ± 47.2 mg/dL), interleukin-6 (32.0 ± 21.5 vs. 43.5 ± 34.3 pg/mL), and high-sensitivity C-reactive protein (5.1 ± 3.7 vs. 9.5 ± 15.6 mg/L) ($P \leq 0.05$). In the intervention group, higher levels of high-density lipoprotein cholesterol (49.2 ± 15.0 vs. 40.4 ± 7.2 mg/dL) and serum total antioxidants (1.9 ± 0.4 vs. 1.4 ± 0.4 mmol/L) were observed ($P < 0.001$). **Conclusion:** A short-term yoga intervention led to reduced glycemia, dyslipidemia, and inflammatory markers and increased antioxidant status in patients with type-2 DM.

Keywords: Breathing exercises, diabetes mellitus, dyslipidemia, inflammation, oxidative stress, yoga

Introduction

The constructive feedback cycle concerning the persistence inflammation, oxidative stress, and development of insulin resistance contributes to diabetes mellitus (DM) and several diabetes-linked complications.^[1] Antidiabetic medication therapy is associated with several adverse effects. Vigorous insulin treatment may also carry an increased risk of atherogenesis.^[2]

Besides being genetically susceptible, the Asian Indian phenotype is also exposed to diverse environmental cues such as an unhealthy diet and sedentary lifestyle.^[3] Lifestyle factors may account for 90% of incident diabetes cases.^[4] Care of persons with diabetes has been influenced by a growing interest in complementary and alternative medicine and one of the methods is yoga practice^[5] and highlighted

in WHO's Global Action Plan on Physical Activity 2018–2030.^[6] The prevention and management of chronic diseases such as diabetes using traditional Indian therapies have been prioritized by the Indian government.^[7]

Studies on yoga intervention in individuals with diabetics have shown considerable heterogeneity in terms of yogic practices and duration and frequency of yoga practice. Most of the studies conducted are short-term studies^[8–13] and studies on the effect of 24-week yoga intervention are scarce or have been performed with lesser frequency of yogic practices. Thus, the present randomized intervention trial was aimed to study the effect of a nonpharmacological intervention, i.e. an integrated approach of traditional yoga therapy for the management of diabetes.

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Methods

Study participants

This study is a prospective randomized clinical trial of yoga intervention conducted over a period of 2 years on 104 participants with type 2 diabetes of either gender in the age group of 30–65 years at government hospital, Rajasthan, India. The study was approved by the institutional ethics committee. This study is registered in Clinical Trial Registry-India (CTRI) number: CTRI/2017/08/009447(Registered on August 22, 2017). Before the commencement of the study, the purpose and procedures were explained to the participants. Written informed consent was obtained from all the study participants.

The diagnosis of diabetes was based on the American Diabetes Association/European Association of Study of Diabetes Criteria.^[14] Participants with infections (acute and chronic), connective tissue disease, bronchial asthma, various viral infections (in the last 1 year), cancer, hemolytic disease, pulmonary tuberculosis, chronic obstructive pulmonary disease, rheumatoid arthritis, psychiatric illness, myalgia, myositis, myopathy, diabetics already practicing yoga or regular physical activity, and seriously ill participants were excluded from the study. All the enrolled participants continued with their diabetic diet and were prescribed, oral hypoglycemic agents. They were not on insulin and were treated with the variation of dose of medicines as advised by the treating physician. The rescue medicines prescribed were oral antidiabetes drugs – metformin, sulfonylureas, gliptins, and others according to physician choice. Patient-level molecules and dosages are not available. A predesigned proforma was used to collect demographic information, lifestyle information, and personal, clinical, and family history.

Randomization of participants

An independent researcher made random allocation using computer-generated randomization process. Randomization sequence was stratified with a 1:1 allocation using random block sizes of 2, 4, and 6. The participants were randomly assigned to one of the two groups, namely the control group without yoga intervention and the intervention group.

A total of 145 participants with diabetes of either gender were screened and out of these, 11 participants did not match the inclusion criteria. Keeping in mind, loss/attrition at 134 participants were enrolled and allocated equally into two study groups, namely the control group without yoga intervention ($n = 67$) and the intervention group ($n = 67$). Twelve participants of the yoga group did not adhere to the yoga intervention schedule, 3 participants were not available for 24-week intervention, and 15 participants were not available in the control group for follow-up. Hence, a total of 104 participants (52 participants for each group) were studied [Figure 1].

Study withdrawal criteria included noncompliance of yoga protocol by participant for more than 60% of time, reporting of any side effects faced that are distressing, if any participant revokes his consent, or develops very high sugar levels during the study.

Yoga intervention

Participants of the intervention group attended approximately 40-min practice of combination of yogasanas and breathing exercises for 24 weeks minimum of 5 days a week. Before start of enrollment of participants, specific yogasana schedule was finalized by trained yoga instructor based on the review of literature and consultation with other yoga experts. The intervention focused on integrating physical and relaxation components which included yogasanas (forward bend, backward bend, and twisted poses), followed by breathing and meditation [Table 1]. All participants of the intervention group were trained for yoga schedules by a single qualified and experienced yoga instructor in yoga laboratory. Printed sheets with instructions, pictures of yogasanas, and recorded DVDs of yoga practices were also handed over to all participants for practice at home. Participants visited yoga centers for yoga practice or performed yoga at home. Compliance to yoga sessions for all participants was monitored telephonically during the study period. No serious adverse effect was reported by any of the participants. The enrolled participants were advised to continue their physical activity, hypoglycemic, and other medicines and the dose of these medicines remained constant throughout the study period.

Anthropometric measurements

Height, weight, waist, and hip circumferences were measured with lightweight clothing and no shoes. Diastolic and systolic blood pressure were measured at baseline and

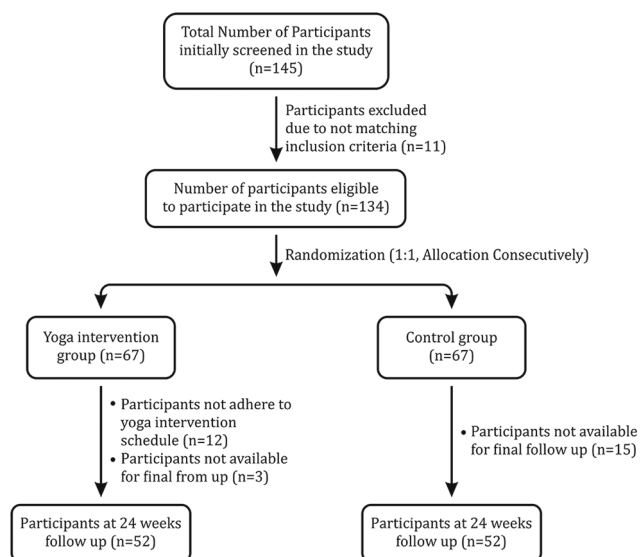


Figure 1: Study flowchart

Table 1: Detailed yoga intervention training protocol

| Yogasana | Execution time | Repetition | Total execution time (min) |
|---|----------------|------------------------|----------------------------|
| Prayer | | Omkar 3 times | 1 |
| Loosening practice (neck movement, shoulder rotation, elbow movements, wrist movements, finger movements, waist rotation, knee rotation, ankle rotation, and toe movements) | 20 s each | - | 3 |
| Asanas (holding time-30 s) | | | |
| Trikonasana | 40 s | 3 | 2 |
| Katichakrasana | 40 s | 3 | 2 |
| Surya Namaskaras | 1.7 min | 3 | 5 |
| Namaskarasana | | | |
| Hastottanasana | | | |
| Padahastanasana | | | |
| Ashwasanchalanasana | | | |
| Parvatasana | | | |
| Ashwasanchalanasana | | | |
| Bhujangasana | | | |
| Parvatasana | | | |
| Ashwasanchalanasana | | | |
| Padahastanasana | | | |
| Hastottanasana | | | |
| Namaskarasana | | | |
| Shavasana | | | 5 |
| Arthamatsyendrasana | 1.5 min | 2 | 3 |
| Pavanamuktasana | 1 min | 3 | 3 |
| Bhujangasana | 1.5 min | 2 | 3 |
| Dhanurasana | 1.5 min | 2 | 3 |
| Padachakrasana | | Clock/anti-clock 15+15 | 2 |
| Relaxation | | | 1 |
| Breathing exercise (One Inhale and One Exhale=One round) | | | |
| Pranayamas | | | |
| Nadi Shodhana (1:4:2) | 20 s | 3 | 1 |
| Ujjayi (1:4:2) | 20 s | 3 | 1 |
| Anulom Vilom (1:4:2) | 20 s | 3 | 1 |
| Bhastrika | 20 s | 3 | 1 |
| Bhramari | 20 s | 3 | 1 |
| Meditation (AUM chanting) | | | 2 |

Source: Saraswati, SS. Asana pranayama mudra bandha. 4th Edition. Munger, Bihar: Yoga publication trust; 2013.^[15]

after 24 weeks of intervention using a standard mercury sphygmomanometer with a cuff adjustment based on arm circumference (Make; Diamond). BMI and waist-hip ratio (WHR) were calculated.^[16]

Biochemical analysis

10–12 h fasting venous blood samples were collected from all participants under aseptic conditions at baseline and after 24 weeks. Blood was analyzed at baseline and at the end of intervention for glucose fasting, postprandial, lipid profile, glycated hemoglobin (HbA_{1c}), creatinine, inflammatory marker high-sensitive C-reactive protein, serum total antioxidants were performed using commercially available reagent kits on fully automated Beckman Coulter analyzer. Interleukin-6 was measured using Diaclone SAS, France; Human ELISA kit (Catalog number: 950.030.096). Complete blood count on automated

cell counter Hemix-5/60 and Horiba medical analyzer. Estimated glomerular filtration rate (ml/min)^[17] and nonhigh-density lipoprotein cholesterol (HDL-C)^[18] were calculated.

Statistical analysis

For each research variable, the mean \pm standard deviation was calculated for both groups. Significance of the difference between means was tested using Student's paired and independent sample "t" tests as per application. The two proportions Z test was used to compare the sex ratio between both the study groups. Pearson's Chi-square test was used to test the significance of the association between categorical variables. Statistical Package for Social Science (SPSS Statistic 22, developed by IBM, Chicago, USA) for Windows was used for data analysis. The results with $P < 0.05$ were considered statistically significant.

Results

The baseline characteristics of study participants are given in Table 2. The mean age of intervention versus control group participants was 52.9 ± 7.9 versus 51.0 ± 7.5 years. The sex ratio in both groups is similar ($P = 0.168$). There is nonsignificant difference in age, gender, BMI, and WHR between the groups at baseline in the present study. Out of 104 study participants, 27 (52%), 20 (38%) were females 25 (48%), and 32 (61.5%) were males in the control group and intervention group, respectively. Nonsignificant difference was observed in smoking habits, alcohol abuse, hypertension, and coronary heart disease [Table 2]. Anthropometric and other biochemical parameters in diabetes participants of the control group and intervention group at baseline, depicting that the grouping was done with no sampling bias, as shown in Table 2.

A highly significant difference was observed in BMI (25.6 ± 2.9 vs. 28.0 ± 3.2 kg/m²); WHR (0.94 ± 0.06 vs. 0.99 ± 0.05); systolic blood pressure (121.2 ± 11.7 vs. 139.3 ± 19.1 mmHg); fasting glucose (142.7 ± 45.3 vs. 175.7 ± 45.4 mg/dL); HbA_{1c} (7.2 ± 1.8 vs. $9.4 \pm 1.9\%$); and HDL-C (49.2 ± 15.0 vs. 40.4 ± 7.2 mg/dL); at 24 weeks,

participants in the intervention versus controls ($P < 0.001$). At 24 weeks, participants in the intervention versus controls had diastolic blood pressure (77.4 ± 7.5 vs. 81.0 ± 8.0 mmHg); postprandial glucose (188.4 ± 43.9 vs. 225.0 ± 65.4 mg/dL); low-density lipoprotein cholesterol (LDL-C) (167.5 ± 38.1 vs. 192.2 ± 51.4 mg/dL); very LDL-C (VLDL-C) (30.0 ± 10.2 vs. 34.8 ± 10.3 mg/dL); and nonHDL-C (136.8 ± 35.3 vs. 158.6 ± 47.2 mg/dL) were observed ($P < 0.05$).

Participants of the intervention group at 24 weeks versus controls at 24 weeks showed significant improved levels of inflammatory markers interleukin-6 (32.0 ± 21.5 vs. 43.5 ± 34.3 pg/mL); high-sensitive C-reactive protein (5.1 ± 3.7 vs. 9.5 ± 15.6 mg/L); Similar trends were observed in total leukocyte counts (8.9 ± 1.6 vs. 9.7 ± 1.1 1000/mm³) and serum total antioxidants (1.91 ± 0.4 vs. 1.4 ± 0.4 mmol/L) ($P < 0.05$) [Table 3].

We also compared within-group changes before and after intervention in the control group and intervention group, data on intragroup comparison are given in the supplementary table. Participants of the control group at 24 weeks versus at baseline showed no significant

Table 2: Baseline characteristics of the study participants

| Variables | Mean±SD | | P* (t-test or Chi-square test) |
|------------------------------------|----------------------|---------------------------|--------------------------------|
| | Control group (n=52) | Intervention group (n=52) | |
| Age (years) | 51.0±7.5 | 52.9±7.9 | 0.214 |
| Men: women | 25:27 | 32:20 | 0.168 |
| Smoker, n (%) | 4 (8) | 3 (6) | 0.695 |
| Alcohol abuse, n (%) | 3 (6) | 2 (4) | 0.667 |
| Hypertension, n (%) | 8 (15.4) | 9 (17.3) | 0.822 |
| Coronary heart disease, n (%) | 2 (3.8) | 01 (1.9) | 0.569 |
| BMI (kg/m ²) | 28.2±3.3 | 27.8±3.1 | 0.621 |
| WHR | 0.99±0.06 | 0.99±0.07 | 0.941 |
| Systolic BP (mmHg) | 142.1±20.1 | 139.5±21.0 | 0.525 |
| Diastolic BP (mmHg) | 83.6±6.7 | 83.8±6.6 | 0.860 |
| Glucose (fasting) (mg/dL) | 183.3±38.9 | 182.0±46.4 | 0.880 |
| Glucose (2 h postprandial) (mg/dL) | 237.1±48.8 | 228.4±42.4 | 0.328 |
| HbA _{1c} (%) | 9.3±1.9 | 9.3±2.1 | 0.852 |
| Total cholesterol (mg/dL) | 198.7±47.7 | 201.4±29.0 | 0.728 |
| Triglycerides (mg/dL) | 181.0±76.3 | 181.7±54.2 | 0.961 |
| HDL-C (mg/dL) | 45.1±14.2 | 42.9±9.59 | 0.373 |
| LDL-C (mg/dL) | 194.9±53.8 | 193.2±31.0 | 0.846 |
| VLDL-C (mg/dL) | 36.0±15.2 | 36.5±10.9 | 0.887 |
| Non-HDL-C (mg/dL) | 153.6±53.1 | 158.4±27.4 | 0.562 |
| Creatinine (mg/dL) | 1.4±0.5 | 1.7±0.9 | 0.115 |
| eGFR (mL/min) | 62.2±15.7 | 67.8±20.4 | 0.123 |
| Hb (g/dL) | 11.7±2.7 | 11.4±2.7 | 0.601 |
| Interleukin-6 (pg/mL) | 42.4±28.9 | 46.6±32.5 | 0.492 |
| High-sensitivity CRP (mg/L) | 8.9±13.7 | 8.29±4.0 | 0.748 |
| Total antioxidants (mmol/L) | 1.4±0.5 | 1.31±0.3 | 0.283 |

*P value, t-test for continuous variables, Chi-square test for categorical variables; Numbers±indicate 1 SD; numbers in parentheses are percent. BP: Blood pressure, CRP: C-reactive protein, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, SD: Standard deviation, Hb: Hemoglobin, BMI: Body mass index, WHR: Waist-hip ratio, HbA_{1c}: Glycated Hb, eGFR: Estimated glomerular filtration rate, VLDL-C: Very LDL-C

Table 3: Anthropometric and biochemical parameters in control and intervention groups at 24 weeks follow-up

| Variables | Mean±SD | | Unpaired <i>t</i> -test, <i>P</i> |
|--|-------------------------------|------------------------------------|--------------------------------------|
| | Control group (<i>n</i> =52) | Intervention group (<i>n</i> =52) | |
| BMI (kg/m ²) | 28.0±3.2 | 25.6±2.9 | <0.001 |
| WHR | 0.99±0.05 | 0.94±0.06 | <0.001 |
| Systolic BP (mmHg) | 139.3±19.1 | 121.2±11.7 | <0.001 |
| Diastolic BP (mmHg) | 81.0±8.0 | 77.4±7.5 | 0.019 |
| Glucose (fasting) (mg/dL) | 175.7±45.4 | 142.7±45.3 | <0.001 |
| Glucose (2 h-postprandial) (mg/dL) | 225.0±65.4 | 188.4±43.9 | 0.001 |
| HbA1c (%) | 9.4±1.9 | 7.2±1.8 | <0.001 |
| Total cholesterol (mg/dL) | 199.0±47.1 | 185.9±29.2 | 0.091 |
| Triglycerides (mg/dL) | 176.9±53.0 | 160.7±46.4 | 0.102 |
| HDL-C (mg/dL) | 40.4±7.2 | 49.2±15.0 | <0.001 |
| LDL-C (mg/dL) | 192.2±51.4 | 167.5±38.1 | 0.006 |
| VLDL-C (mg/dL) | 34.8±10.3 | 30.0±10.2 | 0.020 |
| Non-HDL-C (mg/dL) | 158.6±47.2 | 136.8±35.3 | 0.009 |
| Leukocyte counts (1000/mm ³) | 9.7±1.1 | 8.9±1.6 | 0.008 |
| Interleukin-6 (pg/mL) | 43.5±34.3 | 32.0±21.5 | 0.042 |
| High-sensitive CRP (mg/L) | 9.5±15.6 | 5.1±3.7 | 0.050 |
| Total antioxidants (mmol/L) | 1.4±0.4 | 1.91±0.4 | <0.001 |

Numbers indicate 1 SD. Hb: Hemoglobin, BP: Blood pressure, CRP: C-reactive protein, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, SD: Standard deviation, BMI: Body mass index, WHR: Waist-hip ratio, HbA1c: Glycated Hb, VLDL-C: Very LDL-C

improvements in anthropometric parameters, glucose, lipid profile, total leukocyte counts, interleukin-6, high-sensitive C-reactive protein, and total antioxidant status, however, diastolic blood pressure and HDL cholesterol were significantly decreased ($P < 0.05$).

Participants of the intervention group at 24 weeks versus at baseline showed highly significant improvements in BMI, WHR, systolic blood pressure, diastolic blood pressure, fasting glucose, postprandial glucose, HbA_{1c}, LDL-C, VLDL-C, interleukin-6, and total antioxidant status ($P < 0.001$). A significant improvement was observed in total cholesterol, triglycerides, HDL-C, non-HDL-C, TLC, and high-sensitive C-reactive protein were observed ($P < 0.05$) [Supplementary Table 1].

Discussion

The findings of this 24-week randomized clinical yoga intervention trial in participants with diabetes show a significant improvement in the metabolic changes which included subclinical inflammation, glycemic control, atherogenic dyslipidemia, and oxidative stress in the intervention group.

A systematic review on the effect of diabetes management intervention targeted at South Asian patients with type 2 diabetes reported that using lifestyle modifications and diabetes treatment, total mortality is reduced by 46%, cardiovascular death by 57%, and nonfatal cardiovascular events by 59%.^[19] Majority of the participants of the present study fall under the high-risk category [Table 2]. BMI and WHR were significantly decreased in the intervention group after 24 weeks [Table 3]. Physical

activities such as yoga increase lipolysis and decrease fatty acids in body organs significantly improves lean muscle mass and BMI.^[20] The slow and nonstrenuous activities of yoga positively affect the hypothalamus-pituitary axis response to stress and this is the main basis for the reduction in weight loss.^[21] In the present study, blood pressure was decreased significantly in the intervention group. These are in accordance with other studies evaluated in a meta-analysis.^[22]

After 24 weeks, a significant reduction in glucose and HbA_{1c} levels was achieved in participants of the intervention group. These results are supported by other studies.^[12,19,22-24] Yogasanas practiced in this study, rejuvenate pancreatic cells through alternating abdominal contractions. Relaxations involved in yoga practice, improve blood supply to muscles, and enhances insulin receptor expression in the muscles, causing glucose uptake by muscles.^[20] Finally, these asanas have positive effects on glucose utilization and fat redistribution in DM.^[25]

Abnormalities of all lipoproteins promote atherosclerosis and an increase in plasma lipids impairs insulin activity.^[26] The significant reduction in total cholesterol, triglycerides, LDL-C, non-HDL, and VLDL values coupled with a significant increase in HDL-C in the intervention group after 24 weeks implies improvement in cardiovascular risk profile of diabetic patients. The findings of the present study are comparable with the findings of other studies.^[12,22,23,25,27] No changes in HDL-C after yoga intervention have also been reported.^[28] This improvement in the lipid profile could be due to increased hepatic lipase and lipoprotein lipase at cellular level, which affects the metabolism of

lipoprotein and thus increases the uptake of triglycerides by adipose tissues.^[29]

Significant improvement in antioxidants (mmol/L) in participants with diabetes ($P = 0.000$) after 24 weeks in this study indicates a possible protective effect of yoga and a marked improvement in the overall cellular antioxidant level in yoga group participants. These findings are supported by other studies.^[12,30] A major benefit of nonexhaustive exercise such as yoga is to induce mild oxidative stress that stimulates the expression of certain antioxidant enzymes. This is mediated by the activation of redox-sensitive signaling pathways.^[31]

Inflammation plays a major role in the defense mechanism of the body. In our study, levels of inflammatory markers (interleukin-6 and high-sensitive C-reactive protein) were found to be significantly decreased in the intervention group after 24 weeks [Table 3]. Our results are in accordance with other studies Tolahunase *et al.*^[30] and Pullen *et al.*^[32] Significant changes are observed in total leukocyte count in this study after the yoga intervention. Banerjee *et al.*^[33] reported a significant increase in Hb, platelet count, and decrease in WBC in healthy volunteers after yoga practice. Beneficial effects of yoga on diabetes-related risk profiles appear to occur through two main pathways. First, by reducing the activation and reactivity of the sympathoadrenal system and the hypothalamic–pituitary–adrenal axis and by promoting feelings of well-being, yoga may alleviate the effects of stress and foster multiple positive downstream effects on the neuroendocrine status, metabolic function, and related systemic inflammatory responses. Second, by directly stimulating the vagus nerve, yoga may enhance parasympathetic activity and lead to positive changes in the cardiovagal function, mood, energy state, and related neuroendocrine, metabolic, and inflammatory responses.^[34] Improvement in quality of life with yoga and meditation has been reported in our previous study.^[35]

The results suggest that yoga as a lifestyle therapy can stall or delay the progression of metabolic deterioration in the individual with diabetes with regard to improvement in metabolic parameters in comparison to those diabetic individuals who are solely on standard diabetes management protocol. Our results are supported by the findings of a meta-analysis which highlights that yoga is beneficial in type 2 DM and is having a supportive role to play along with pharmacotherapy in ensuring good metabolic control.^[36]

Strength and limitations of the study

The strength of this randomized control trial is that it is a 24-week intervention study which includes an experimental as well as a control group. The schedule of integrated yogasanas was developed as a systematic approach for type 2 diabetics. The duration (24 weeks) and frequency of

practice (5 days in a week) of our intervention are longer than many other yoga interventions^[8-13] which also had considerable methodological limitations.

There are few limitations in this study. First, participants were not blinded to yoga intervention. Second, compliance of yoga was based on feedback on adherence to the home-based yoga practice of participants as it was not feasible to make them perform yoga daily at the yoga center of the institute for 24 weeks. Finally, although all participants were advised standard lifestyle measures, their diet was not followed and their heart rate was not measured. Insulin levels were not measured due to limited funding issues. Despite these limitations, the findings of this study may account for positive outcomes by adopting yoga as a lifestyle intervention in participants with diabetes.

Conclusion

Yoga intervention practiced 5 days a week for a period of 24 weeks has shown a beneficial impact on BMI, glycemic control, lipid profile, inflammation, and antioxidants in participants with diabetes which lays a scientific foundation regarding the advantages of yoga in diabetes. Individuals with diabetes should be encouraged to fully embrace yoga as the results of the study highlight its cardioprotective potential and also efficacy as a lifestyle intervention in the management of DM.

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Ethical statement

The study was approved by the institutional Ethics committee of RUHS College of Medical Sciences, Jaipur, Rajasthan, India. [DCG (I)ECR/762/Inst/RJ/2015].

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Conflicts of interest

There are no conflicts of interest.

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Supplementary Table 1: Anthropometric and biochemical parameters at baseline and after 24 weeks of intervention in control and intervention groups

| Variables | Control group, mean±SD | | | Intervention group, mean±SD | | |
|--|------------------------|------------|------------|-----------------------------|------------|------------|
| | Baseline | 24 weeks | <i>P</i> * | Baseline | 24 weeks | <i>P</i> * |
| BMI (kg/m ²) | 28.2±3.3 | 28.0±3.2 | 0.515 | 27.8±3.1 | 25.6±2.9 | <0.001 |
| WHR | 0.99±0.06 | 0.99±0.05 | 0.876 | 0.99±0.07 | 0.94±0.06 | <0.001 |
| Systolic BP (mmHg) | 142.1±20.1 | 139.3±19.1 | 0.101 | 139.5±21.0 | 121.2±11.7 | <0.001 |
| Diastolic BP (mmHg) | 83.6±6.7 | 81.0±8.0 | 0.039 | 83.8±6.6 | 77.4±7.5 | <0.001 |
| Glucose (fasting) (mg/dL) | 183.3±38.9 | 175.7±45.4 | 0.243 | 182.0±46.4 | 142.7±45.3 | <0.001 |
| Glucose (2 h postprandial) (mg/dL) | 237.1±48.8 | 225.0±65.4 | 0.158 | 228.4±42.4 | 188.4±43.9 | <0.001 |
| HbA1c (%) | 9.3±1.9 | 9.4±1.9 | 0.953 | 9.3±2.1 | 7.2±1.8 | <0.001 |
| Total cholesterol (mg/dL) | 198.7±47.7 | 199.0±47.1 | 0.961 | 201.4±29.0 | 185.9±29.2 | 0.008 |
| Triglycerides (mg/dL) | 181.0±76.3 | 176.9±53.0 | 0.484 | 181.7±54.2 | 160.7±46.4 | 0.003 |
| HDL-C (mg/dL) | 45.1±14.2 | 40.4±7.2 | 0.036 | 42.9±9.59 | 49.2±15.0 | 0.017 |
| LDL-C (mg/dL) | 194.9±53.8 | 192.2±51.4 | 0.712 | 193.2±31.0 | 167.5±38.1 | <0.001 |
| VLDL-C (mg/dL) | 36.0±15.2 | 34.8±10.3 | 0.260 | 36.5±10.9 | 30.0±10.2 | <0.001 |
| Non-HDL-C (mg/dL) | 153.6±53.1 | 158.6±47.2 | 0.413 | 158.4±27.4 | 136.8±35.3 | 0.001 |
| Leukocyte counts (1000/mm ³) | 9.4±1.6 | 9.7±1.1 | 0.146 | 8.50±2.1 | 8.9±1.6 | 0.021 |
| Interleukin-6 (pg/mL) | 42.4±28.9 | 43.5±34.3 | 0.811 | 46.6±32.5 | 32.0±21.5 | <0.001 |
| High-sensitive CRP (mg/L) | 8.9±13.7 | 9.5±15.6 | 0.334 | 8.3±4.0 | 5.1±3.7 | 0.001 |
| Total antioxidants (mmol/L) | 1.4±0.5 | 1.4±0.4 | 0.537 | 1.31±0.3 | 1.91±0.4 | <0.001 |

**P* value, unpaired *t*-test for continuous variables; Numbers± indicate 1 SD. BP: Blood pressure, CRP: C-reactive protein, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, SD: Standard deviation, BMI: Body mass index, Hb: Hemoglobin, WHR: Waist-hip ratio, HbA1c: Glycated Hb, VLDL-C: Very LDL-C