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RESEARCH ARTICLE

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# Effect of turmeric on glycemic status, lipid profile, hs-CRP, and total antioxidant capacity in hyperlipidemic type 2 diabetes mellitus patients

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Diabetes mellitus is the most common metabolic disorder worldwide. This study examined the effect of turmeric supplementation on glycemic status, lipid profile, hs-CRP and total antioxidant capacity in hyperlipidemic type 2 diabetic patients. In this double-blind, randomized clinical trial, 80 hyperlipidemic type 2 diabetic patients were divided into turmeric (2,100 mg powdered rhizome of turmeric daily) and placebo groups for 8 weeks. Body weight, fasting plasma glucose, hemoglobin A1c (HbA1c), serum insulin, triglyceride (TG), total cholesterol, low density lypoprotein cholesterol (LDL-c), high density lypoprotein cholesterol, apolipoprotein A1, apolipoprotein B, high sensitivity C-reactive protein (hs-CRP), and total antioxidant capacity were measured before and after intervention. Statistical analysis was carried out using paired and independent *t* and chi-square tests. Seventy five patients completed the study. The turmeric group showed significant decreases in body weight, TG, and LDL-c compared with baseline ($p$ value < 0.05). Body mass index, TG, and total cholesterol decreased significantly in the turmeric group compared with the placebo group ($p$ value < 0.05). No significant changes were observed in other parameters between the two groups after intervention ($p$ value < 0.05). Turmeric improved some fractions of lipid profile and decreased body weight in hyperlipidemic patients with type 2 diabetes. It had no significant effect on glycemic status, hs-CRP, and total antioxidant capacity in these patients.

**KEYWORDS**

glycemic status, hs-CRP, lipid profile, total antioxidant capacity, turmeric, type 2 diabetes

**Abbreviations:** BMI, Body mass index; COX, Cyclooxigenase; FBG, Fasting blood glucose; HbA1C, Hemoglobin A1c; HDL-c, High-density lipoprotein; HOMA-IR, Homeostatic model assessment of insulin resistance; hs-CRP, High-sensitivity C-reactive Protein; IL, Interleukin; IPAQ, International Physical Activity Questionnaire; LDL-c, Low-density lipoprotein; PG, Prostaglandin; TAC, Total antioxidant capacity; TG, Triglyceride

  

## 1 | INTRODUCTION

Diabetes mellitus is one of the most common metabolic diseases all around the world. It is due to insulin deficiency or inadequate response to insulin, which leads to elevated blood glucose and disturbance in the metabolism of carbohydrates, fats, and proteins (American Diabetes Association, 2014). It is expected that the prevalence of type 2 diabetes among adults increases from 285 million patients in the year 2010 to 438 million patients in the year 2030. More than 60% of the diabetic patients live in Asia. There are more than 4 million type 2 diabetic patients in Iran (Haque et al., 2011; Peimani et al., 2010; Shaw, Sicree, & Zimmet, 2010).

Insulin resistance and impaired insulin function can lead to dyslipidemia in diabetic patients. Increased triglyceride (TG) and low density lypoprotein cholesterol (LDL-c) and decreased high density lypoprotein cholesterol (HDL-c) are the characteristics of dyslipidemia in type 2 diabetic patients. Dyslipidemia, inflammation, and oxidative stress are the major risk factors of atherosclerosis and cardiovascular diseases (Marion et al., 2002; Tan, 2004).

Today, many researchers consider the use of spices and plant-derived compounds to reduce insulin resistance, oxidative stress, and chronic inflammation related to diabetes (Marion et al., 2002). Turmeric (*Curcuma longa*) is a member of the ginger family (Zingiberaceae) and has been used as a spice in Asian countries since ancient times. Its main active components are yellow pigments of curcuminoides that forms 3%–5% of turmeric, which composed of curcumin, demethoxy curcumin, and bisdemethoxy curcumin. Curcumin is the most important compound of turmeric that has the most impact on health (Arablou & Kolahdouz-Mohammadi, 2018; Srinivasan, 2005). Recent researches on turmeric showed that the plant has multiple pharmacological actions. Because curcumin prevents the formation of oxygen free radicals and has antioxidant properties, it may be effective in reducing diabetes progression and complications (De Man, Castro Cabezas, Van Barlingen, Erkelens, & De Bruin, 1996; Zahid Ashraf, Hussain, & Fahim, 2005). In vitro and animal model studies have shown the antioxidant, anti-inflammatory, blood lipids and sugar-reducing properties of turmeric (Bengmark & Gil, 2009; Healthcare, 2007; Ray, Chansouria, & Hemalatha, 2010). In Minpei kuroda et al. study, 0.2 or 1.0 g/100 g diet (260 and 1,500 mg/kg body weight) of turmeric extract in the diet of diabetic mice for 4 weeks prevented the blood glucose elevation and improved insulin resistance (Kuroda et al., 2005). In the study of Ho et al., 200, or 500 mg/kg body weight curcumin ethanol extract in the diet of obese mice for 9 weeks led to weight loss and improved lipid profile (Ho et al., 2012).

Because turmeric is used widely in Iranian traditional medicine and as a common spice in Iranian foods, and to the best of our knowledge, there had been no clinical study on the effect of turmeric on metabolic status of hyperlipidemic patients with type 2 diabetes and also, previous related studies have reported some inconsistent results; the present study was conducted to evaluate the effect of turmeric powder on glycemic status, lipid profile, high sensitivity C-reactive protein (hs-CRP), and total antioxidant capacity in hyperlipidemic type 2 diabetic patients.

## 2 | MATERIALS AND METHODS

### 2.1 | Study design

This is a randomized, double-blind clinical trial that is approved by the medical ethics committee of Tehran University of Medical Sciences, which conforms to the provision of Helsinki in 1995 (as revised in 2000) and registered in the Iranian Registry of Clinical Trials (IRCT NO 201204162602).

### 2.2 | Study population

The study population included 30 to 70 year-old hyperlipidemic type 2 diabetic patients who were referred to the Endocrinology and Metabolism Research Center in Firoozgar Hospital of Iran University of Medical Sciences. The inclusion criteria were type 2 diabetic patients with fasting blood sugar < 200 mg/dl, hemoglobin A1c (HbA1C) > 6%, TG > 150 mg/dl, or LDL-c > 100 mg/dl, body mass index (BMI) between 20 and 35 kg/m$^2$, no insulin therapy, and no use of antioxidants, multivitamin, or polyphenols supplements for the last 3 months prior to the study. The exclusion criteria were any changes in physical activity, diet, and drug intake during the period of the study, fasting blood sugar > 200 mg/dl or LDL-c > 160 mg/dl, acute heart disease, renal or liver failure, thyroid disease, severe gastrointestinal disease, gastric ulcers, biliary stones, pregnancy, or lactation. The eligible patients with physician endorsement were invited and all of them gave written informed consent to participate in the study.

### 2.3 | Sample size calculation

The sample size was calculated using one of the similar previous studies (Khadem Haghighian, Farsad Naimi, Pourghassem Gargari, Ali-Asgharzadeh, & Nemati, 2011), considering the fasting blood glucose as the main variable, and using the following formula with 80% power and 95% confidence interval.

$$n = 2\,\delta^2_{\mathrm{d}}\,\left(Z_{1-\alpha/2}-Z_{1-\beta}\right)^2/(d_1-d_2)^2$$

$$\left(Z_{1-\alpha/2}=1.96 \text{ and } Z_{1-\beta}=0.84\right).$$

The calculated sample size was 35 participants in each group. Considering the anticipation of % 10 drop-outs, the sample size was estimated to 40 participants in each group.

### 2.4 | Randomization

Eighty eligible patients were randomly divided into two groups: the intervention ($n$ = 40) and placebo ($n$ = 40) groups according to the block randomization method (block size 4).

## 2.5 | Intervention

The intervention group received 2,100 mg turmeric powder (three 700 mg turmeric capsules after main meals) and the placebo group received 2,100 mg corn starch flour as placebo (three 700 mg capsules after main meals) daily for 8 weeks. The participants were asked not to make any special changes in their diet and physical activity and report any changes in medications during the intervention period and the only intervention was the turmeric or placebo supplementation.

## 2.6 | Blinding

In order to blind the intervention, turmeric supplements and placebos were prepared in quite similar appearance and were given to the participants by someone other than the researcher. Neither researcher nor the patients were aware of turmeric or placebo group members.

## 2.7 | Supplement preparation

The rhizome of turmeric was purchased from a local herb store in Tehran and approved by an herbalist and deposited with voucher number T457. The rhizomes were cleaned, dried, and grounded into powder. On the basis of previous human studies, turmeric consumption in the amount of 1–3 g/day did not show any side effects (Bengmark & Gil, 2009; Healthcare, 2007; Khajehdehi et al., 2012). As a result, we chose the 2,100 mg dose (three 700 mg capsules). The powder was filled in gelatin capsules. Each capsule contains 700 mg of turmeric powder. Placebo capsules contained 700 mg of corn starch flour prepared in similar shape and color.

In order to increase the compliance, participants were called on the phone during the intervention to be reminded to receive the supplements. Supplements were given monthly, and at the end of each month, people were asked to bring empty boxes of their supplements. The compliance was calculated by counting the number of consumed supplements during the intervention, which if less than 80%, the participant was excluded from the study.

## 2.8 | Analytical procedures

At baseline, demographic data were obtained by interview. To control the confounding effects of dietary intake, 24-hr recall questionnaire (1 regular day) and food diary (2 days: 1 regular day and 1 weekend day) were obtained and data were analyzed by Nutritionist IV software (version 3.5.2, The Hearst Corporation, San Bruno, CA). Physical activity was evaluated by International Physical Activity Questionnaire (IPAQ). Body weight was measured in the fasting state with light clothing and without shoes using Seca scale (Seca, Hamburg, Germany); and height was measured without shoes using a stadiometer attached to the scale. BMI was calculated by dividing the weight in kilogram by the square of height in meter.

Blood samples (10 ml) were taken in 12-hr fasting state at the beginning and after 8 weeks of intervention. After separation of serum, blood parameters were measured. These parameters include: total cholesterol (cholesterol oxidase method, Pars Azmun kit, Iran), fasting plasma sugar (glucose oxidase method, Pars Azmun kit, Iran), TG (Gop-Pap method, Pars Azmun kit, Iran), LDL-c and HDL-c (direct method, Pars Azmun kit, Iran), insulin (chemiluminescence immunoassay, monobind Inc, Insulin AccuBind, Lake Forest, CA, USA), and HbA1C (Ion exchange chromatography method, $DS_5$, Britain). Insulin resistance was calculated as HOMA score using the US formula:

HOMA-IR = fasting blood glucose (mg/dl) × fasting serum insulin ($\mu$U/ml)/405.

Apolipoprotein A1 and apolipoprotein B were measured by the immune turbidometery method (Cobasintegra 400, a Roche Company, Germany). hs-CRP and total antioxidant capacity were measured using ELISA and colorimetric method (both LDN kit, Germany), respectively.

## 2.9 | Statistical analysis

Statistical analysis was performed using SPSS software (version16; SPSS Inc., Chicago, IL). The Kolmogorov–Smirnov test was used to determine data compliance with the normal distribution. Quantitative variables were compared between groups at baseline and at the end of the study using an independent *t* test. Quantitative variables before and after treatment within each group were compared using paired *t* test. Qualitative variables were analyzed by the chi squared test. Also, considering that the BMI changes may affect glycemia, lipid profile, and inflammatory markers, the ANCONA test was used to assess the probable confounding effect of BMI on study variables. All analyses were performed on participants who completed the study duration (39 in turmeric and 36 in the placebo group). All values are reported based on Mean ± *SD*. $p$ value < 0.05 was considered as the statistical significance level.

# 3 | RESULTS

During the study period, five participants were excluded due to changes in medication dose, the need for insulin therapy, travel, and unwillingness to cooperate; and finally the study ended with 75 patients (39 patients in the intervention group and 36 in the placebo group; Diagram 1). None of the participants in the turmeric or in the placebo groups reported any side effects of supplementation and all the participants had the compliance of more than 80%.

Baseline characteristics of participants are presented in Table 1. Comparisons showed no significant differences in age, sex, medications, and duration of the diabetes between the two groups at baseline ($p$ value > 0.05). Also, there were no significant differences in physical activity and diet components between the two groups at baseline and the end ($p$ value > 0.05) (Data not shown).

Table 2 shows the anthropometric indices and glycemic status of the participants. Anthropometric indices have no significant differences between the two groups at baseline ($p$ value > 0.05). Body weight reduced significantly in the intervention group during the study ($p$ value < 0.001). Between group differences in body weight at the

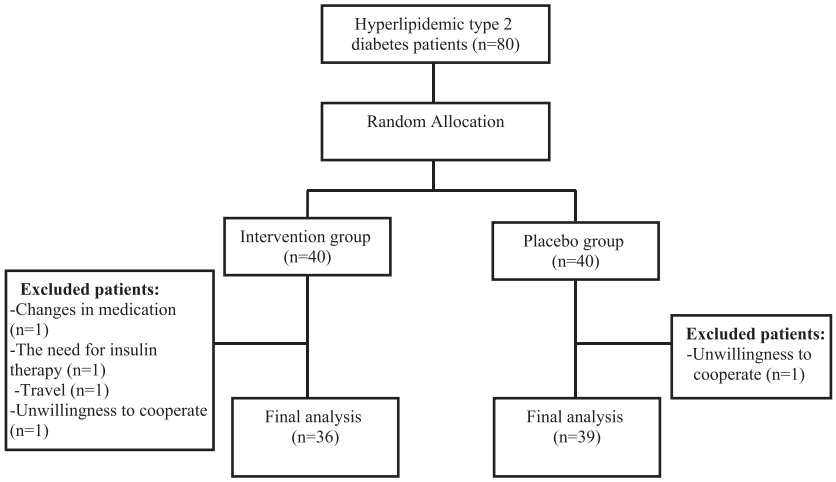


**DIAGRAM 1** Flowchart of study participants

**TABLE 1** Baseline characteristics of study participants

| Characteristics | Intervention group (*n* = 39) | Placebo group (*n* = 36) | *p* value |
|---|---|---|---|
| Age (year) | 54.76 ± 6.00 | 55.66 ± 8.64 | 0.60[a] |
| Sex | | | |
| Female (%) | 20 (51.0) | 19 (52.8) | >0.99[b] |
| Male (%) | 19 (48.7) | 17 (47.2) | |
| Blood sugar-lowering agents | | | |
| Metformin (%) | 18 (46.2) | 17 (47.2) | 0.46[b] |
| Glibenclamide (%) | 3 (7.7) | 4 (11.1) | |
| Metformin+ | 16 (41.1) | 13 (36.1) | |
| Glibenclamide (%) | 2 (5.1) | 2 (5.6) | |
| Gliclazide (%) | | | |
| Blood lipid-lowering agents | | | |
| Atorvastatin (%) | 30 (76.9) | 21 (58.3) | 0.11[b] |
| Cholestyramine (%) | 8 (20.5) | 10 (27.8) | |
| Fenofibrate or Gemfibrozil (%) | 1 (2.6) | 5 (13.9) | |
| Duration of diabetes | | | |
| >5 Years (%) | 20 (51.3) | 19 (52.8) | 0.45[b] |
| 5–10 Years (%) | 19 (48.7) | 17 (47.2) | |

*Note*. Data of age is presented as mean ± *SD*. *p* value < 0.05 is significant.

[a]Independent *t* test.

[b]Chi-squared test.

beginning and the end of the study were not statistically significant (*p* value > 0.05). In patients who received turmeric, a significant decrease in BMI was observed at the end of the study compared with the beginning (*p* value < 0.001). Also mean differences of BMI in intervention group reduced significantly compared with the placebo group (*p* value = 0.001).

As Table 2 shows, the mean fasting plasma glucose level, HbA1c, insulin, and HOMA-IR reduced in the turmeric group compared with the placebo group, but these reductions were not statistically significant. Also, before and after changes in these parameters were not statistically significant (*p* value > 0.05). The mean of HbA1c increased significantly in the placebo group during the study (*p* value = 0.03), whereas in the intervention group such an increase was not observed (*p* value > 0.05).

The lipid profile before and after the intervention is summarized in Table 3. TG and total cholesterol in the placebo group increased significantly (*p* value = 0.006 and *p* value = 0.001 respectively), whereas in the intervention group, TG decreased markedly (*p* value < 0.001) and total cholesterol had no significant changes (*p* value > 0.05) during the study period. The mean differences of TG and total cholesterol between the two groups were statistically significant (*p* value < 0.001 and *p* value = 0.004, respectively).

HDL-c decreased in two groups after intervention, but this reduction was significant only in the placebo group (*p* value = 0.02). However, the mean differences of HDL-c were not statistically significant (*p* value > 0.05). After intervention, LDL-c decreased significantly in turmeric group compared with baseline (*p* value = 0.009). Mean differences of LDL-c between two groups were not statistically significant (*p* value = 0.050). Mean differences of apolipoprotein A1 and apolipoprotein B were not significant between the two groups (*p* value > 0.05).

Table 4 shows changes of hs-CRP and total antioxidant capacity in patients before and after the intervention. Results showed that changes of hs-CRP and total antioxidant capacity (TAC) were not statistically significant between the two groups and within each group before and after the intervention (*p* value > 0.05).

Moreover, the ANCOVA test analysis showed that changes in BMI had no confounding effect on the changes of other study variables (*p* value > 0.05).

## 4 | DISCUSSION

In the present study, consumption of 2,100 mg of turmeric for 8 weeks caused significant reduction in body weight and BMI in the intervention group compared with baseline. Also, mean differences of BMI in the turmeric group reduced significantly compared with the placebo group.

**TABLE 2** Anthropometric indices and glycemic status of the participants before and after the intervention

| Variable | Intervention group ($n$ = 39) | $p$ value* | Placebo group ($n$ = 36) | $p$ value* | $p$ value** |
|---|---|---|---|---|---|
| Body weight (kg) | | | | | |
| Before | 76.86 ± 10.36 | <0.001 | 74.56 ± 17.04 | 0.40 | 0.58 |
| After | 75.05 ± 9.96 | | 76.68 ± 14.36 | | 0.57 |
| Differences | −1.80 ± 1.70 | | 2.12 ± 15.01 | | 0.10 |
| BMI ($kg/m^2$) | | | | | |
| Before | 28.98 ± 3.68 | <0.001 | 28.82 ± 4.96 | 0.29 | 0.88 |
| After | 28.26 ± 3.45 | | 28.68 ± 4.86 | | 0.67 |
| Differences | −0.71 ± 0.63 | | 0.14 ± 0.80 | | 0.001 |
| FBG (mg/dl) | | | | | |
| Before | 133.79 ± 25.60 | 0.57 | 129.91 ± 32.98 | 0.059 | 0.57 |
| After | 131.64 ± 28.33 | | 139.41 ± 41.57 | 0.35 | |
| Differences | −2.15 ± 23.76 | | 9.50 ± 29.23 | 0.06 | |
| HbA1C% | | | | | |
| Before | 7.06 ± 1.01 | 0.90 | 6.79 ± 1.08 | 0.03 | 0.26 |
| After | 7.04 ± 0.98 | | 7.28 ± 1.59 | | 0.45 |
| Differences | −0.01 ± 0.97 | | 0.48 ± 1.36 | | 0.07 |
| Insulin (mIU/L) | | | | | |
| Before | 7.29 ± 4.92 | 0.75 | 7.29 ± 4.77 | 0.26 | 0.99 |
| After | 7.11 ± 5.17 | | 8.15 ± 5.72 | | 0.41 |
| Differences | −0.18 ± 3.64 | | 0.86 ± 4.56 | | 0.27 |
| HOMA-IR | | | | | |
| Before | 2.42 ± 1.73 | 0.26 | 2.24 ± 1.48 | 0.18 | 0.63 |
| After | 2.21 ± 1.43 | | 2.69 ± 2.02 | | 0.23 |
| Differences | −0.21 ± 1.18 | | 0.44 ± 1.96 | | 0.07 |

*Note*. Data are presented as mean ± *SD*. $p$ value < 0.05 is significant. BMI: body mass index; FBG: fasting blood glucose; HbA1C: hemoglobin A1c; HOMA-IR: homeostatic model assessment of insulin resistance.

*$p$ value within group comparison (paired $t$ test). **$p$ value between group comparison (independent $t$ test).

Previous studies on the effect of turmeric on anthropometric indices have shown inconsistent findings. Although, Sukandar et al. reported that ethanolic extract of garlic and turmeric reduced BMI significantly in type 2 diabetic patients with dyslipidemia (Sukandar et al., 2010), however two clinical trials on overweight/obese women with systemic inflammation and dyslipidemia showed that supplementation with 2.8 and 4 g turmeric powder for 4 and 8 weeks, respectively, had no significant effect on body weight and BMI compared with the placebo group (Kabaran & Atakan, 2018; Nieman, Cialdella-Kam, Knab, & Andrew-Shanely, 2012).

Animal studies also showed that body weight and adipose tissue decreased with curcumin treatment (Ho et al., 2012). It seems that turmeric can increase lipolysis via protein kinase-A pathway (Ho et al., 2012). Also, curcumin increases fatty acids oxidation. Meanwhile, increasing basal metabolic rate and decreasing the level of inflammatory cytokines can lead to reduced adipose tissue and weight loss (Aggarwal, 2010; Alappat & Awad, 2010).

With regard to issue of glycemia in the present study, turmeric reduced fasting plasma glucose, HbA1c, serum insulin, and insulin resistance index (HOMA), but these reductions were not statistically significant compared with the placebo group. To date, limited human and animal studies have been carried out on the effect of turmeric on glycemia, which reported some incompatible results (Sukandar et al., 2010, Wickenberg, Ingemansson, & Hlebowicz, 2010, Rungseesantivanon, Thenchaisri, Ruangvejvorachai, & Patumraj, 2010; Pongchaidecha, Lailerd, Boonprasert, & Chattipakorn, 2009). Perhaps the reasons for incompatible findings in glycemic status in different studies are the use of full turmeric powder or curcumin, the dose of treatment, differences in study design, and duration. Some experimental studies indicate that curcumin is an effective anti-diabetic agent. The role of curcumin in glucose homeostasis is utilized through activating glycolysis, preventing gluconeogenesis, and decreasing hepatic lipid metabolism. Also, it improves insulin sensitivity by reducing insulin resistance (Alappat & Awad, 2010; Kuroda et al., 2005) and stimulation of pancreatic β cell function (Wickenberg et al., 2010).

In the present study, turmeric caused significant decrease in serum triglycerides and LDL-c in hyperlipidemic patients with type 2 diabetes in 8 weeks and also prevented the increase in serum total cholesterol. There was no significant difference in mean change in HDL-c, and the decrease which was observed in the intervention group was not statistically significant unlike that of the placebo group.

**TABLE 3** Lipid profile of the participants before and after the intervention

| Variable | Intervention group (*n* = 39) | *p* value* | Placebo group (*n* = 36) | *p* value* | *p* value** |
|---|---|---|---|---|---|
| TG (mg/dl) | | | | | |
| Before | 181.56 ± 79.79 | <0.001 | 164.05 ± 81.19 | 0.006 | 0.35 |
| After | 141.74 ± 52.02 | | 197.05 ± 96.98 | | 0.004 |
| Differences | −39.82 ± 58.80 | | 33.0 ± 67.79 | | <0.001 |
| Total cholesterol (mg/dl) | | | | | |
| Before | 148.85 ± 36.11 | 0.75 | 155.36 ± 36.27 | 0.001 | 0.43 |
| After | 149.82 ± 35.67 | | 176.88 ± 37.58 | | 0.002 |
| Differences | 0.97.03 ± 19.21 | | 21.52 ± 37.09 | | 0.004 |
| HDL-c (mg/dl) | | | | | |
| Before | 38.79 ± 10.30 | 0.06 | 44.63 ± 10.66 | 0.02 | 0.19 |
| After | 37.07 ± 9.12 | | 42.11 ± 9.39 | | 0.02 |
| Differences | −1.71 ± 5.61 | | −2.52 ± 21.46 | | 0.55 |
| LDL-c (mg/dl) | | | | | |
| Before | 82.56 ± 20.99 | 0.009 | 86.61 ± 21.99 | 0.58 | 0.41 |
| After | 75.23 ± 18.84 | | 89.05 ± 21.46 | | 0.004 |
| Differences | −7.33 ± 16.75 | | 2.44 ± 24.76 | | 0.050 |
| Apolipoprotein A1 | | | | | |
| Before | 136.64 ± 21.57 | 0.24 | 145.77 ± 20.55 | 0.02 | 0.06 |
| After | 138.95 ± 23.68 | | 151.40 ± 20.23 | | 0.01 |
| Differences | 2.31 ± 12.25 | | 5.63 ± 14.72 | | 0.29 |
| Apolipoprotein B | | | | | |
| Before | 89.47 ± 23.35 | 0.47 | 90.16 ± 20.05 | 0.18 | 0.90 |
| After | 87.88 ± 21.90 | | 96.06 ± 27.77 | | 0.16 |
| Differences | −1.59 ± 13.90 | | 5.90 ± 26.08 | | 0.13 |

*Note*. Data are presented as mean ± SD. *p* value < 0.05 is significant. TG: triglyceride; HDL-c: high-density lipoprotein; LDL-c: low-density lipoprotein.
**p* value within group comparison (paired *t* test). ***p* value between group comparison (independent *t* test).

**TABLE 4** hs-CRP and total antioxidant capacity of the participants before and after the intervention

| Variable | Intervention group (*n* = 39) | P-value* | Placebo group (*n* = 36) | P-value* | P-value** |
|---|---|---|---|---|---|
| hs-CRP (mg/L) | | | | | |
| Before | 2.65 ± 2.94 | 0.91 | 2.76 ± 2.72 | 0.43 | 0.86 |
| After | 2.59 ± 2.90 | | 2.52 ± 2.40 | | 0.90 |
| Differences | −0.06 ± 3.38 | | −0.24 ± 1.81 | | 0.77 |
| TAC (mmol/L) | | | | | |
| Before | 1.66 ± 0.45 | 0.14 | 1.71 ± 0.40 | 0.67 | 0.63 |
| After | 1.81 ± 0.43 | | 1.68 ± 0.38 | | 0.17 |
| Differences | 0.14 ± 0.63 | | −0.03 ± 0.08 | | 0.37 |

Data are presented as mean ± SD. *p* value < 0.05 is significant. hs-CRP: high-sensitivity C-reactive protein; TAC: total antioxidant capacity.
**p* value within group comparison (paired *t* test).
***p* value between group comparison (independent *t* test).

Although, apolipoprotein A1 increased in both groups after the intervention, however this increase was statistically significant only in the placebo group, and the mean differences of this parameter were not significant between the two groups after the intervention. The changes of apolipoprotein B were not significant between the two groups.

The study of Sukandar et al., on type 2 diabetic patients with dyslipidemia, showed that ethanolic extract of garlic and turmeric significantly decreased serum TG, total cholesterol, and LDL-c. Also, HDL-c was increased significantly with the dose of 2.4 g (Sukandar et al., 2010). The result of this study is consistent with our study with regard to the effect of turmeric in reducing lipid profile, but in the case

of HDL-c the result is inconsistent. This contradiction probably can be justified in this way: In the Sukandar et al. study, patients were under intensive diet and exercise for 2 weeks prior to the study, then the patients whose blood lipids and glucose were not improved, supplemented with garlic and turmeric. However, in our study, diet and physical activity of patients had no significant differences at the beginning and the end of the study. The effects of diet and exercise on serum HDL-c were proved by several studies (Afzalaghaiee et al., 2010; Pasdary, Alghasi, Rashidi, & Rezai, 2012). In addition, the patients in our study were receiving blood sugar reducing agents and statins that obviously affected lipid pattern especially in the placebo group. In the present study, turmeric prevented more reduction in HDL-c in the intervention group compared with the placebo group. In another trial on acute coronary syndrome patients, turmeric and curcumin decreased total cholesterol and LDL-c and increased HDL-c (Alwi et al., 2008). Furthermore, in most of the animal studies, curcumin reduced TG, total cholesterol, LDL-c, and free fatty acids and increased HDL-c and apolipoprotein A1 (El-Moselhy, Taye, Sharkawi, El-Sisi, & Ahmed, 2011; Ho et al., 2012; Jang et al., 2008; Pongchaidecha et al., 2009).

Researchers believe that the possible mechanism that turmeric improves dyslipidemia, is that it can elevate cholesterol catabolism by increasing liver cholesterol 7-hydroxylase enzyme activity and inhibiting the synthesis of cholesterol by suppression of the HMG-CoA reductase enzyme. Also, curcumin affects LDL-c receptors and inhibits absorption of dietary cholesterol (Sukandar et al., 2010; Zahid Ashraf et al., 2005). To date, various studies showed that curcumin can prevent fatty acid synthase enzyme activity and increases fatty acids β-oxidation, through which reduces fat storage and regulates lipid metabolism (Zahid Ashraf et al., 2005).

Furthermore, the present study showed that turmeric consumption could not significantly change hs-CRP and total antioxidant capacity in the intervention group. Evidence represents the fundamental role of inflammation and oxidative stress in cardiovascular disease initiation and progression (Lee et al., 2009; Mahajan et al., 2009; Pradhan, Manson, Rifai, Buring, & Ridker, 2001). Serum CRP is a very sensitive marker of inflammation in hyperglycemia, insulin resistance, and dyslipidemia. Several studies have suggested that antioxidant and polyphenols-rich diets can prevent the production and function of proinflammatory molecules such as CRP (Kowluru & Kanwar, 2007; Song, Manson, Buring, Sesso, & Liu, 2005). Many studies have proven the anti-inflammatory properties of turmeric via inhibiting the synthesis of proinflammatory cytokines and mediators such as COX-2, prostaglandins, and leucotriens (Aghaee Burashan, Ilkhanipour, Hashemi, & Farrokhi, 2008; Rungseesantivanon, Thengchaisri, Ruangvejvorachai, & Patumraj, 2010; Yun, Jialal, & Devaraj, 2011).

However, to the best of our knowledge, the present study is the first to examine the effect of turmeric on hs-CRP and TAC in hyperlipidemic type 2 diabetic patients. Previous clinical trials on the effect of turmeric supplementation on different disorders have reported some inconsistent findings. It seems that the observed incoherence was due to the differences in metabolic profile of the patients, the dose of turmeric supplements, intervention duration, and sample sizes (Amin, Islam, Anilac, & Gilania, 2015; Nazarali, Shadkam, & Shemshaki, 2015; Nieman et al., 2012; Pakfetrat, Basiri, Malekmakan, & Roozbeh, 2014).

In several animal models, turmeric and curcumin reduced inflammatory markers including CRP and ceruloplasmin, and proinflammatory cytokines such as IL-1β and IL-6 and increased anti-inflammatory cytokines such as IL-10 and IL-4 (Aghaee Burashan et al., 2008; Nemmar, Subramaniyan, & Ali, 2012; Ramadan, Al-Kahtani, & El-Sayed, 2011). The reason of no significant changes of hs-CRP levels in our study may be attributed to the normal value of this parameter at baseline (2.65 ± 2.94 mg/L) and the need for the longer period of treatment. One of the causes of normal value of hs-CRP in patients before the intervention might be receiving statin drugs by patients. It is reported that statins can reduce inflammation (Asghari, Hosseinzadeh Attar, Mohajeri Tehrani, & Sehhat, 2011).

According to evidence, these components also, increased significantly TAC and antioxidant enzymes such as glutathione peroxidase and glutathione and reduced significantly lipid peroxidation and malondialdehyde in animal studies (Arun & Nalini, 2002; Durgaprasad, Pai, Alvres, & Namitha, 2005; Hosseinzadeh & Dabidi Roshan, 2011; Madkor, Mansour, & Ramadan, 2011).

The researchers believe that turmeric can reduce lipid peroxidation by maintaining antioxidant enzymes activity such as superoxide dismutase, catalase, and glutathione peroxidase at high level. However, turmeric can destroy oxygen free radicals, which have a major role in lipid peroxidation (Vahdatpour & Mashayekhi, 2009).

In the present study, turmeric supplementation of diabetic patients with dyslipidemia could not significantly change the glycemic status, hs-CRP, and TAC. This is probably because of insufficient time course.

The present study had some limitations. We could not assess the outcomes at longer time course. It would be better if we could perform the trial with the longer intervention duration and also more participants. Therefore, further studies with longer time period, larger sample size, and comparing the effect of turmeric powder with curcumin in hyperlipidemic patients with type 2 diabetes are suggested.

## 5 | CONCLUSION

In conclusion, this study showed that daily intake of 2,100 mg turmeric powder for 8 weeks had no effect on glycemic status, hs-CRP, and TAC, but reduced BMI, serum TG, and LDL-c in hyperlipidemic patients with type 2 diabetes; and likely can reduce diabetes complications, atherosclerosis, and overweight in these patients as an adjunct therapy.

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## CONFLICT OF INTEREST

The authors declare no conflict of interest. The study was funded by Tehran and Iran University of Medical Sciences.

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## SUPPORTING INFORMATION

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