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Regulation of Serum Lipid Profile, Glucose, Insulin, and Adiponectin in Obese Diabetic Women Under Diet Therapy: A Randomized Clinical Controlled Study

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Abstract

Background: Obesity is the main cause of insulin resistance and type 2 diabetes mellitus (T2DM). Diet therapy is the cornerstone in the management of obesity and T2DM.
Objectives: We evaluated the effects of calorie-restricted diet therapy on the circulating level of the serum lipid profile, glucose, insulin, and adiponectin in obese women with T2DM.
Materials and Methods: This randomized clinical controlled trial was performed for 10 weeks on 30 eligible obese T2DM women distributed to control (n = 15) and diet therapy (n = 15) groups. Demographic, nutritional, anthropometric, and laboratory data were obtained before and after the study. Data were analyzed by SPSS vs.15 and Nutritionist IV.
Results: In addition to anthropometric measurements, diet therapy independently improved fasting blood sugar (P = 0.024, -69.37 to -5.57 mg/dL), 2-h postprandial blood sugar (P = 0.007, -123.34 to -22.3 mg/dL), serum total cholesterol (P = 0.005, -46.48 to -9.72 mg/dL), serum alanine transaminase (P = 0.001, -8.91 to -3.18 U/L), and increased circulating adiponectin (P = 0.038, 0.01 to 0.47 μg/mL).
Conclusions: Improvement of biomarkers of insulin sensitivity, including adiponectin and lipid metabolism, is an important therapeutic effect of medical nutrition therapy in obese patients with T2DM.

Keywords: Diet Therapy, T2DM, Obesity

1. Background

Communities have been faced with the increasing burden of obesity (body mass index (BMI) $\geq$ 30 kg/m^2), which can be attributed to around half of the worldwide incidence of type 2 diabetes mellitus (T2DM). Diabetes is more prevalent among Middle Eastern populations compared to those living in other countries. Approximately 8% and 17% of adults living in Iran are predicted to have T2DM and impaired fasting glucose, respectively (1). The rising prevalence of diabetes and its complications in tandem with overweight and obesity shows the correlative and causative links between obesity and insulin resistance (1). Moreover, these statistics indicate that more compressive approaches need to be recognized in the prevention and treatment of obesity. The American diabetes association (ADA) has reported the critical role of nutritional factors and has emphasized the importance of medical nutrition therapy in the prevention and management of T2DM and its complications (2). Despite the ADA's recommendations (2, 3), only a few studies (4, 5) have examined the impact of standard dietary macronutrient distribution on glucose and lipid metabolism biomarkers in obese patients suffering from insulin resistance. Most studies have assessed the effects of some unusual diets, such as very low calories, or dietary plans with uncommon percentages of macronutrients without considering the patient's lifestyle and dietary habits and without planning a personalized diet in obese and diabetic patients (6, 7). To study the independent effect of diet therapy on dysmetabolic parameters in obese patients with T2DM, we should take into account all parameters that have effects on metabolism homeostasis, including lifestyle, anthropometric, biochemical, and nutritional factors. We hypothesized that a personalized diet based on a patient's dietary habits and requirements could improve dysmetabolic parameters in obese patients with T2DM.

2. Objectives

We evaluated the effects of calorie-restricted diet therapy on the circulating level of the serum lipid profile, glucose, insulin, and adiponectin in obese women with T2DM.

3. Materials and Methods

3.1. Study Population

The sample size was calculated giving consideration to LDL-c outcomes (8) on the basis of Pocock's sample size formula with a power of 80% and a 95% confidence interval, taking into account 30% for probable loss. The work was carried out in compliance with CONSORT guidelines. The format of this randomized controlled clinical trial, which ran from February 2014 to January 2015, was approved by ethics committee of Tabriz University of Medical Sciences (code no 92162) and by the Iranian registry of clinical trials (IRCT) (code no IRCT2014011416223N1). Informed written consent was obtained from all volunteers. Thirty-two patients who met the inclusion criteria were chosen from 55 initial volunteers from the diabetes clinic of Emdadi hospital, Zanjan University of Medical Sciences, Zanjan, Iran. Block randomization (1:1) with a block size of 4 was done by the statistician of the study using RAS software. Inclusion criteria were women who were obese (BMI $\geq$ 30 kg/m^2), 25 - 60 years old, and with T2DM for a minimum of 2 years. Of the 32 selected women, 16 were allocated to the treatment group and 16 to the control group. The T2DM diagnosis of the patients was made based on world health organization (WHO) criteria (9). Patients were under treatment with gliburide/glucophage tablets. The exclusion criteria were patient pregnancy; lactation; diabetic ketoacidosis; presentation of ketonuria; history of cardiovascular, liver failure, renal diseases, and malabsorption disorders; BMI $\geq$ 45 kg/m^2; any change in medications throughout the study, incomplete questionnaires; unwillingness to give blood samples; following special dietary plans or use of dietary supplements within the last year; having weight changes or a history of hormone therapies within the last 3 months; insulin requirements; and having T1DM.

3.2. Dietary Intake

In order to obtain a habitual diet, a valid 168-item semi-quantitative food frequency questionnaire (10) was completed by a trained dietitian at the beginning of the study. Dietary records for two weekdays and one weekend were filled out by each participant. The portion sizes of consumed food were converted to grams using household measures (11) and were then coded and analyzed for their energy content and other nutrients, using Nutritionist IV software, which was designed for Iranian food.

3.3. Diet Planning

In the diabetic treatment group, the personalized total energy expenditure was calculated by subtracting 700 kcal from that obtained using the standard equation considering the patient's current weight and physical activity coefficient (12). The habitual diets were designed by a trained dietitian with 17%, 53%, and 30% of energy as proteins, carbohydrates, and fat, respectively, based on the concepts of low glycemic index (GI) and glycemic load (GL). Patients followed the diets for 10 weeks; participants were asked not to alter their medication, although medications between two diabetic groups were adjusted in line with the blocked randomization. The control group continued their habitual diet. In the follow-up that occurred every 5 weeks, a 3-day dietary record was obtained to estimate the subjects' adherence to the study.

3.4. Demographic, Clinical, and Anthropometric Measurements

Demographic questionnaires, completed as a baseline, included information on detailed medications, education level, job, menopause status, stress quality and severity, quantity of sleep, physical activity, history of abortion or pregnancy, marital status, family history of obesity, diabetes, hypertension, and cardiovascular disease (CVD). Clinical and anthropometric questionnaires were completed at the start and at the 5th and 10th weeks of the study. Systolic and diastolic blood pressures (BPs) were measured in duplicate with an upper arm blood pressure monitor (Beurer BM35; Beurer GmbH; Germany). Patients rested several minutes prior to the BP measurement and were seated with their arm comfortably resting on a table. Duplicate BP readings were obtained on the right arm with an interval of at least 5 minutes between readings. For analysis, the average of the duplicate values was used. Body weight, total body fat (%), and visceral fat (%) were measured using a calibrated and validated body composition monitor scale (Omeron, Seoul, Korea) while the subjects were fasting and wearing soft indoor outfits without shoes. Height was measured using a portable stadiometer (Seca, UK) to the nearest 0.1 cm with the subjects stretching to their maximum height and their head positioned in the Frankfort plane. BMI was calculated as weight in kilograms divided by the square of height in meters. Waist circumference (WC), at the point between the lowest rib and the superior border of the right iliac crest, and hip circumference, (HC) at the widest point around the greater trochanters, were measured to the nearest 0.5 cm with a measuring tape (Seca) directly on the individual's skin. The waist-to-hip ratio (WHR) was calculated as WC divided by HC. Skinfold thickness was measured to the nearest 1 mm at the following points: subscapular, biceps, triceps, and

supra-iliac, using a Harpenden skinfold caliper (Germany) directly on the individual's skin. All measurements and questionnaires were collected by the same trained dietitians.

3.5. Biochemical Measurements

Before and after 10 weeks of study, 10-cc fasting blood samples were taken in 7.5% K3-EDTA tubes (Vacutainer System; Becton Dickinson, UK). After inverting five times, the tubes were centrifuged for 10 minutes at 13,000 $\times$ g (Eppendorf 5702; US). The following methods were used for biochemical assessments: GOD/PAP for both fasting blood sugar (FBS) and 2-hour postprandial blood sugar (2h PPBS), GPO-PAP for serum triglycerides (TG), enzymatic endpoint for serum total cholesterol (TC), enzymatic clearance evaluations for both direct high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) and photometric evaluations for both serum alanine transaminase (ALT) and aspartate transaminase (AST). All tests were performed using a Randox Laboratories kit (UK) (Hitachi 902; Roche Diagnostics, USA).The homeostasis model assessment of insulin resistance (HOMA-IR) and β-cell activity (HOMA-β) were calculated before the study and 10 weeks after the study (13).

Biochemical assessments were done using human enzyme-linked immunosorbent assay (ELISA) kits as follows: serum glucagon (Cusabio Pharmaceuticals, Hubei Province, China; sensitivity 4.3 pg/mL, intra-CV < 8%, and inter-CV < 10%), serum adiponectin (Biovendor Pharmaceuticals, GmbH, Germany; sensitivity 26 ng/mL, intra-CV 4.9%, and inter-CV 6.7%), fasting and 2-hour postprandial insulin (Monobind Pharmaceuticals, CA, USA; sensitivity 0.182 μIU/ mL, intra-CV 5.5%, and inter-CV 9.1%), and serum TNF-α (eBioscience Pharmaceuticals, Vienna, Austria; sensitivity 2.3 pg/mL, intra-CV 6%, and inter-CV 7.4%). All analyses were done by a trained laboratory specialist in duplicate under the same conditions at the biotechnology centralized laboratory, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

3.6. Statistical Analysis

Statistical analysis was performed using SPSS software, version 15.0 (SPSS, USA). Kolmogorov–Smirnov descriptive analysis and chi-square testing were used to test for normal distribution and homogeneity of variances. Quantitative variables were matched with the chi-square test. Log10-transformation was done as necessary. The significance of the total energy and macronutrient changes were examined with repeated analysis of variance (ANOVA). The independent t-test or Mann-Whitney U test and the paired t-test or Wilcoxon test were used for group comparison. To adjust for multiple variables on intervention, univariate analysis of covariance was used. Descriptive statistics were computed using mean $\pm$ standard deviation (SD). For all analyses, a two-sided P value of < 0.05 was deemed statistically significant.

4. Results

4.1. Disposition, Demographic, and Clinical Characteristics of the Diabetic Patients

During the study, one patient from each of the control and treatment groups was lost to follow-up due to a leg fracture and travel, respectively. Thirty diabetic women completed the study (Figure 1). Participants were frequency matched for quantitative variables of education level, job, menopause status, stress, quality and quantity of sleep, physical activity, history of abortion or pregnancy, marital status, and family history of obesity, diabetes, hypertension, and cardiovascular diseases ($P > 0.05$). There were no differences in the number of subjects with these characteristics between the two diabetic groups. Demographic and clinical characteristics of participants are shown in Table 1.

4.2. Total Energy and Macronutrient Intake of the Treatment Group

We tested the normal distribution, homogeneity, and sphericity after 10 weeks on the diet therapy and found a significant reduction of caloric, carbohydrate, and total fat intake in the treatment group (Table 2). In the intervention group, there were significant reductions of energy (MD $\pm$ SD = -286.752 $\pm$ 47.18, $P < 0.001$), dietary carbohydrate (-52.08 $\pm$ 11.08, $P < 0.001$), and dietary fat (-9.87 $\pm$ 3.24, $P = 0.011$) intake during the study.

4.3. Independent Effect of Calorie-Restricted Diet on Glucose and Lipid Metabolism Biomarkers

Table 3 shows the independent effects of the calorie-restricted diet on the systolic BP, anthropometric measurements, FBS, 2h PPBS, and adiponectin in the treatment group.

5. Discussion

Total energy, fat and dietary carbohydrate intakes decreased significantly in the diabetic treatment group during our study. After 10 weeks, the mean calorie restriction in the treatment group was about 227 Kcal/day (Table 2). Assuming a 1.7 kg weight loss in the diabetic treatment group over 10 weeks and a 7 - 9 Kcal energy density per gram of body weight (12), patients adhered to the individual diet

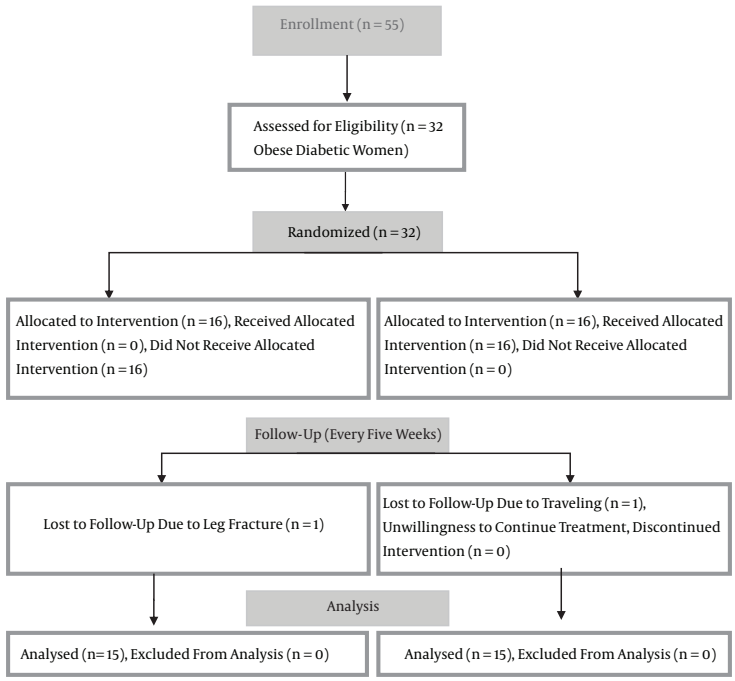

Figure 1. Study's CONSORT Workflow

about 32% - 35% (222 - 227 Kcal/day) of the time. In general, adherence to therapeutic programs is a multidisciplinary issue, and WHO statistics show that the adherence of diabetic patients to therapeutic programs is usually less than 50% (14). In this study, the diet therapy with moderate fat and carbohydrate restrictions was associated with improved dysmetabolic parameters, including BP, visceral and subcutaneous fat deposits, FBS, 2h PPBS, serum total cholesterol, serum ALT, and circulating adiponectin.

In 2015, the ADA considered 140 mg/dL, 180 mg/dL, and 100 mg/dL as the maximum limit values of FBS, 2h PPBS, and LDL-c, respectively, in diabetic patients with cardiovascular risk factors (15). Interestingly, we showed that the diet therapy could improve excess FBS, 2h PPBS, and LDL-c by 60%, 83.7%, and 112%, respectively, in obese women with T2DM. By taking 25 kg/m^2 for BMI and 90 cm for WC (16) as the cut-off points for Iranian women and mean weight (81.28 kg) and mean WC (108.38 cm) (Table 1) in the treatment group before the study, our diet therapy reduced about 8% and 40% of the excess weight and WC, respectively, in obese women with T2DM. In agreement with a previous study (17), our significant improvements in FBS and 2h PPBS in the treatment group might be attributed to decreased hepatic glucose output and insulin resistance due to reduced subscapular and visceral fats (Table 3). However, in contrast with earlier animal and human reports (18, 19), we did not see any significant effect of diet therapy on the HOMA-IR score. Adiponectin is an insulin-sensitizing adipocytokine that paradoxically decreases in obese and diabetic patients (20). Our results showed a significant increase in circulating levels of adiponectin in obese patients under the diet therapy, confirming some earlier reports (18, 21, 22). The increased circulating adiponectin level could partially explain one important clinical effect of diet therapy on glycemic control in patients with T2DM. In general, these findings showed that a moderate calorie-restricted diet therapy can improve insulin secretion and resistance in T2DM and the responsible pathologic pathways in obesity (23). TNF-α is also an adipocytokine; it is known to be a cardiovascular risk factor in T2DM and independently causes inflammation and insulin resistance in obese patients (24). In contrast to some studies that reported a significant reduction in TNF-a in obese patients under a calorie-restricted diet therapy (25, 26) and in agreement with others (27, 28), we did not observe any significant effect of the calorie-restricted diet on circulating TNF-α in patients.

The high incidences of hypertension and hyperlipidemia along with impaired glycemic controlthree important risk factors of CVD in diabetes have been reported in obesity. In addition to the hypoglycemic effect from the diet therapy, we found significant improvements in sys-

Table 1. Demographic and Clinical Characteristics of Participants[a]

	Control (n = 15)		Treatment (n = 15)		P Value[b]	
	Baseline	After the Study	Baseline	After the Study	Baseline	After the Study
Age, y	49.63 ± 9.57	49.63 ± 9.57	49.28 ± 7.75	49.28 ± 7.75	0.920	-
Diabetes medications, n/d	3.15 ± 1.45	3.15 ± 1.45	3.71 ± 1.63	3.71 ± 1.63	0.764	-
Duration of diabetes, y	6.45 ± 4.27	6.45 ± 4.27	8.00 ± 4.64	8.00 ± 4.64	0.401	-
SBP, mm Hg	13.34 ± 1.99	13.17 ± 1.56	14.05 ± 1.80	11.92 ± 3.24[c]	0.364	0.261
DBP, mm Hg	8.17 ± 0.95	7.94 ± 0.80[c]	10.00 ± 2.32	8.10 ± 0.95[d]	0.023[d]	0.680
Weight, kg	82.80 ± 13.45	82.60 ± 14.18	81.28 ± 10.76	79.94 ± 11.65[c]	0.757	0.626
BMI, kg/m^2	34.57 ± 5.62	34.45 ± 5.97	32.45 ± 2.34	31.86 ± 2.40[c]	0.212	0.180
Visceral fat, level	11.09 ± 2.66	11.54 ± 2.84	10.33 ± 1.43	9.83 ± 1.46[d]	0.296	0.727
WC, cm	109.97 ± 14.09	111.50 ± 15.05	108.36 ± 8.61	104.17 ± 7.99[c]	0.727	0.154
HC, cm	114.91 ± 12.27	115.41 ± 12.05	113.08 ± 9.35	109.96 ± 8.99[d]	0.676	0.230
WHR	0.95 ± 0.06	0.96 ± 0.07	0.96 ± 0.05	0.94 ± 0.02	0.909	0.459
Biceps fat, cm	2.75 ± 0.49	2.63 ± 0.58	2.77 ± 0.54	2.22 ± 0.36[b]	0.928	0.053
Triceps fat, cm	2.83 ± 0.33	3.03 ± 0.45	3.02 ± 0.44	2.68 ± 0.40[e]	0.263	0.063
Suprailiac fat, cm	5.55 ± 0.95	5.48 ± 1.086	5.65 ± 1.18	4.32 ± 1.00[e]	0.819	0.014[c]
Suescapular fat, cm	4.80 ± 1.30	5.07 ± 1.32[c]	5.19 ± 1.28	4.49 ± 0.95[f]	0.468	0.223
FBS, mg/dL	182.64 ± 69.40	172.82 ± 64.68	202.43 ± 72.60	148.50 ± 72.05[e]	0.497	0.417
2h PPBS, mg/dL	254.91 ± 119.39	257.36 ± 100.00	267.14 ± 88.92	192.92 ± 77.90[e]	0.771	0.098
Cholesterol, mg/dL	178.55 ± 36.21	190.36 ± 48.92	207.20 ± 24.68	203.00 ± 25.23	0.046	0.427
HDL-C, mg/dL	36.54 ± 5.75	38.14 ± 6.78	41.42 ± 8.49	39.66 ± 9.25	0.116	0.660
LDL-C, mg/dL	102.09 ± 43.11	125.83 ± 20.13	124.00 ± 22.36	109.91 ± 53.25	0.113	0.346
AST, U/L	23.00 ± 8.96	23.54 ± 6.77	24.64 ± 18.10	20.84 ± 8.57	0.786	0.408
ALT, U/L	23.90 ± 9.72	25.36 ± 7.07	25.78 ± 10.3	20.33 ± 6.22[f]	0.648	0.084
Biceps fat, cm	2.75 ± 0.49	2.63 ± 0.58	2.77 ± 0.54	2.22 ± 0.36[e]	0.928	0.053
Triceps fat, cm	2.83 ± 0.33	3.03 ± 0.45	3.02 ± 0.44	2.68 ± 0.40[e]	0.263	0.063
Suprailiac fat, cm	5.55 ± 0.95	5.48 ± 1.086	5.65 ± 1.18	4.32 ± 1.00[e]	0.819	0.014[c]
Subscapular fat, cm	4.80 ± 1.30	5.07 ± 1.32[f]	5.19 ± 1.28	4.49 ± 0.95[f]	0.468	0.223
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Abbreviations: ALT, alanine transaminase; AST, aspatate transaminase; BMI, body mass index; DBP, diastolic blood pressure; FBS, fasting blood sugar; HC, hip circumference; HDL-C, high-density lipoprotein; HOMA-IR or β, homeostatic model assessment of insulin resistance or β-cell function; L, logarithm; LDL-C, low-density lipoprotein; SBP, systolic blood pressure; TNF-α, tumor necrosis factor alpha; WC, waist circumference; WHR, waist to hip ratio; 2h PPSB, 2-h postprandial blood sugar.

[a] Values are expressed as mean ± SD. pi, p value from independent t-test for nonlogarithmic and exact p value from the Mann-Whitney U test for logarithmic values;

[b] P value from independent t-test.

[c] BP < 0.01.

[d] P < 0.05 and for paired comparisons before versus after study (paired t-test).

[e] P < 0.05.

[f] P < 0.01 for paired comparisons before versus after study (paired t-test for nonlogarithmic and exact P value Wilcoxon test for logarithmic values).

tolic BP and total cholesterol in obese women with T2DM (Table 3). In agreement with these findings, one randomized controlled trial and a recent meta-analysis (17, 29) showed significant improvements in fasting glucose and total cholesterol in T2DM patients under low-carbohydrate diets. Interestingly, the prevalence of non-alcoholic fatty liver disease (NAFLD) in patients with combined T2DM and obesity has been reported as 100% (30). A high prevalence of NAFLD in T2DM is associated with a higher prevalence of CVD, hepatocellular carcinoma, and a greater burden of diabetic complications (31). We demonstrated that a moderate low-calorie diet with management of dietary GI/GL was adequate for a significant reduction of ALT (-6 mg/dL, $P \leq 0.01$) in obese diabetic patients.

A number of reported contradictions in different studies can be attributed to different intervention times, amount of caloric restriction, and the subject's physical activity. Our observations showed that to achieve good con-

Table 2. Changes in Dietary Macronutrients During the Study[a,b]

Measures	Control (n = 15)	Treatment (n = 15)
Energy intake, baseline, Kcal/d	1787.64 ± 318.59	1882.27 ± 149.94
Energy intake, 5th week, Kcal/d	1848.00 ± 284.89	1714.10 ± 142.88
Energy intake, 10th week, Kcal/d	1787.1 ± 322.05	1595.1 ± 110.58
P Value[c]	0.445	0.001[d]
Carbohydrate intake, baseline, g/day	225.91 ± 50.53	231.48 ± 27.34
Carbohydrate intake, 5th week, g/d	229.92 ± 48.76	198.21 ± 34.44
Carbohydrate intake, 10th week, g/d	214.71 ± 43.24	179.40 ± 33.08
P Value	0.396	0.001[d]
Protein intake, baseline, g/d	51.71 ± 10.68	52.87 ± 6.14
Protein intake, 5th week, g/d	50.65 ± 10.76	52.27 ± 5.81
Protein intake, 10th week, g/d	54.17 ± 10.87	55.80 ± 9.30
P Value	0. 473	0.215
Fat intake, baseline, g/d	75.23 ± 14.08	82.76 ± 10.87
Fat intake, 5th week, g/d	80.63 ± 20.03	78.26 ± 13.07
Fat intake, 10th week, g/d	79.13 ± 21.20	72.88 ± 10.13
P Value	0.326	0.019[d]

[a] Results of the repeated measure ANOVA.
[b] Values are mean ± SD.
[c] P value from sphericity-assumed test or Greenhouse-Geisser test have been reported based on the results of Mauchly's test.
[d] Difference is significant ($P \leq 0.05$).

trol of glucose and lipid metabolism in obese diabetic patients, it is not necessary to adopt overwhelming dietary plans or diets inconsistent with a patient's eating habits. A moderate calorie restriction of dietary carbohydrates and fats may be a safe strategy for the treatment and control of insulin resistance in obese patients. Our study is a novel, comprehensive, human study that matched most patients' confounding characteristics, including sex, geographic region, lifestyle, dietary habits, physical activity, medications, and anthropometric and biochemical indices of metabolism homeostasis, for reporting the independent therapeutic effects of diet therapy in obese patients with T2DM. The limitation of this study is that we could not continue the intervention for more than 10 weeks under stable conditions because of the importance of constant medicines throughout the study.

Acknowledgments

This report is based on a database from a Ph.D. thesis and had ethics approval from the ethical committee of Tabriz University of Medical Sciences (code no. 92162) and acceptance in the Iranian registry of clinical trials (IRCT; code IRCT2014011416223N1) before the enrollment of the first patients. The authors appreciate all the participants who patiently stated their experiences.

Footnotes

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Author's Contribution: Somayeh Mohammadi developed the original idea and protocol and wrote the abstract and the manuscript. Seyed Rafie Arefhosseini and Mehrangiz Ebrahimi-Mameghani contributed to the development of the protocol and coordination of the study. Mohammad Asghari Jafarabadi participated in the analysis and interpretation of data and in the design of the study. Zarin sharifnia carried out biochemical measurements and acquisition of biochemical data.

Table 3. Effects of the Diet Therapy on Glucose and Lipid Metabolic Biomarkers[a]

	F	P Value	MD (95% CI)
SBP, mmHg	11.388	0.003[b]	-2.76 (-4.470 - -1.05)
Weight, kg	6.677	0.018[b]	-1.73 (-3.13 - -0.33)
BMI, kg/m²	3.367	0.082	-0.56 (-1.20 - 0.08)
Waist circumference, cm	20.524	0.001[b]	-7.31 (-10.70 - -3.93)
Hip circumference, cm	9.348	0.006 [b]	- 4.36 (-7.35 - -1.37)
Biceps fat, mm	5.466	0.030[b]	–0.39 (-0.74 - -0.04)
Triceps fat, mm	9.737	0.006[b]	–0.44 (-0.74 - -0.14)
Subscapular fat, mm	5.290	0.030[b]	–0.74 (-1.42 - -0.06)
Super-iliac fat, mm	10.00	0.005[b]	- 0.91 (-1.52 - -0.31)
Visceral adipose tissue, %	17.874	0.001[b]	-1.13 (-1.70 - -0.57)
FBS, mg/dL	6.044	0.024[b]	-37.47 (-69.37 - -5.57)
2h PPBS	9.128	0.007[b]	-72.86 (-123.34 - -22.38)
Total cholesterol, mg/dL	10.241	0.005[b]	-28.10 (-46.48 - -9.72)
ALT, mg/dL	19.540	0.001[b]	-6.05 (-8.91 - -3.18)
L-Adiponectin, μg/mL	4.985	0.038[b]	0.24 (0.01 - 0.47)

Abbreviations: ALT, alanine transaminase; BMI, body mass index; ; FBS, fasting blood sugar; L, logarithm; SBP, systolic blood pressure; 2h PPSB, 2-h postprandial blood sugar.
[a]Results of the analysis covariance (ANCOVA) test.
[b]The mean difference (MD) is significant at the 0.05 level; aadjusted for the difference in caloric intake.

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