Comparative effects of carbohydrate versus fat restriction on metabolic profiles, biomarkers of inflammation and oxidative stress in overweight patients with Type 2 diabetic and coronary heart disease: A randomized clinical trial

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Original Article

Abstract

BACKGROUND: This study was conducted to establish the comparative effects of carbohydrate versus fat restriction on metabolic indices in Type 2 diabetic (T2D) patients with coronary heart disease (CHD).

METHODS: This randomized, clinical trial was done among 56 overweight persons with T2D and CHD aged 40-85 years old. The patients were randomly allocated to take either a high-carbohydrate (HC) diet (60-65% carbohydrates and 20-25% fats) (n = 28) or a restricted carbohydrate (RC) diet (43-49% carbohydrate and 36-40% fats) (n = 28) for 8 weeks to determine metabolic status.

RESULTS: After 8 weeks of treatment, RC diet decreased fasting plasma glucose (FPG) (−11.5 ± 28.3 vs. +7.0 ± 26.9 mg/dl, P = 0.010) and high-sensitivity C-reactive protein (hs-CRP) (−564.3 ± 1280.1 vs. +286.1 ± 1789.2 ng/ml, P = 0.040) compared with a HC diet. Moreover, compared with a HC diet, RC diet increased total antioxidant capacity (TAC) (+274.8 ± 111.5 vs. +20.2 ± 82.5 mmol/l, P < 0.001) and glutathione (GSH) levels (+51.6 ± 111.5 vs. −32.6 ± 88.5 µmol/l, P = 0.003). No significant alterations between the two groups were found in terms of their effect on other metabolic profiles.

CONCLUSION: RC diet in overweight T2D with CHD had beneficial effects on FPG, hs-CRP, TAC, and GSH values.

Keywords: Carbohydrate Restriction, Metabolic Status, Type 2 Diabetes Mellitus, Coronary Heart Disease, Obesity

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Introduction

Type 2 diabetes mellitus (T2DM) is a metabolic disease and is estimated to reach 439 million persons worldwide in 2030.[1] Prior studies have exhibited that the prevalence of obesity and T2DM in people with coronary heart disease (CHD) exceeds that of the general population.[2] Different factors have been involved in the progression of T2DM and CHD such as little glycemic control and dyslipidemia.[3,4] In addition, low-grade inflammation resulting from free radicals and reactive oxygen species (ROS) may help to the expansion of metabolic complexity in diabetic vascular disease.[5-7]

However, it remains unknown whether favorable effects of restricted carbohydrate (RC) diets are mediated through changes in metabolic profiles, the inflammatory process, and endothelial dysfunction. Some studies have revealed that carbohydrate limitation has a more favorable effect on aspects of the metabolic syndrome (MeTs) than a low-fat diet.[8,9] In a study by Parillo et al.[10] was observed that high-monounsaturated-fat/low-carbohydrate diet compared with low-monounsaturated-fat/high-carbohydrate (HC) diet decreased postprandial glucose, insulin and triglycerides values among patients with T2DM for 15 days, but unchanged other lipid profiles. Likewise, low-carbohydrate (20%) than low-fat diet significantly improved the inflammatory state in T2DM after 6 months.[11] However, in a study, a HC diet significantly increased insulin and triglycerides concentrations by 8% and 13%, respectively, and lowered high-density lipoprotein (HDL)-cholesterol

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by 6% compared with the low-carbohydrate diet.[12] In addition, few studies have also shown that acute ingestion of carbohydrate clearly induces ROS, inflammation and oxidative stress.[13,14]

To our knowledge, information on the effects of RC versus HC intake on metabolic status in overweight T2DM persons with CHD is limited. This research, therefore, was done to establish the effects of RC intake and its replacement with unsaturated fats on metabolic parameters in these persons.

Materials and Methods

This treatment was a randomized clinical trial, which was done at the Cardiology Clinic of KUMS, Kashan, Iran, between November 2015 and January 2016. At baseline, people were matched according to age, body mass index (BMI), gender, and the dosage and kind of drugs. Since all people were overweight, both diets were designed to be calorie limited (350-700 kcal less than the computed energy). The macronutrient composition of the HC diet was equal with Iranian usual diets.[15] Indeed, in the RC diet, 15-20% of the energy from carbohydrates was replaced by nonhydrogenated vegetable oils. The protein content of both diets was 14-17% of the total energy. To increase compliance, persons were given a portion list of food groups and solely educated about the goals of each phase as well as the portion list. To take nutrient intakes of people according to 3-day food records, we applied Nutritionist IV software (First Databank, San Bruno, CA). Physical activity was described as metabolic equivalents (METs).[16]

In total, 56 patients were randomly divided into two groups: Group A (HC diet; 15 females and 13 males: n = 28) received 60-65% carbohydrates and 20-25% fats and Group B (RC diet; 15 females and 13 males: n = 28) received 43-49% carbohydrate and 36-40% fats for 8 weeks. Inclusion criteria were overweight patients aged 40-85 years old, BMI ≥ 25, having T2DM and CHD. Diagnosis of T2D and CHD was done based on the American Diabetes Association[17] and the American Heart Association,[18] respectively. Exclusion criteria were consuming antiobesity medications within the last 3 months, having an acute myocardial infarction and/or a cardiac surgery within the last 3 months and a major renal or liver failure.

This intervention was confirmed by the Research Ethics Committee of KUMS (reference number IR.Kaums.REC.1394.96) and was registered in the Iranian registry of clinical trials (http://www.irct.ir: IRCT201601025623N61).

Weight and height were quantified at week 0 and week 8 at the cardiology clinic.

About 10 ml fasting blood samples were collected at week 0 and week 8. Fasting plasma glucose (FPG) and lipid parameters were established with enzymatic kits (Pars Azmun, Tehran, Iran). Insulin values were quantified using enzyme-linked immunosorbent assay (ELISA) kit (DiaMetra, Milano, Italy). Indices of insulin metabolism were calculated according to the existing formulas.[19] High-sensitivity C-reactive protein (hs-CRP) was assessed by the commercial ELISA kit. The nitric oxide (NO) using Griess method,[20] total antioxidant capacity (TAC) by Benzie and Strain[21] method, total glutathione (GSH) using the method of Beutler and Gelbart[22] and malondialdehyde (MDA) were determined by spectrophotometric method.[23]

Based on a prior study,[24] we used 2.5 as standard deviation (SD) and 2.1 as the change in mean (d) of homeostatic model assessment insulin resistance (HOMA-IR). Therefore, we needed 24 persons in each group and assuming 4 dropouts in each group; the final sample size was reached to be 28 people.

To establish the normal distribution of indices, the Kolmogorov–Smirnov was utilized. Results of normally distributed markers as mean ± SDs and non-normally distributed markers as median (Q1, Q3) were reported. The intention-to-treat (ITT) analysis of the primary study endpoint was applied for all of the randomly allocated participants with method of the last-observation-carried-forward method.[25] To establish within-group differences (pre- and post-treatment), we used paired-samples t-tests. Pearson chi-square test was used for comparison of categorical markers. To determine the effects of RC diet intake on metabolic parameters, we applied independent samples Student's t-test. To assess the effects of some confounders, we adjusted all analyses using ANCOVA test.

Results

In the RC group, 4 people and in the HC group, 4 people were withdrawn (Figure 1). However, all 56 people were contained in the final analysis using ITT principle.

Mean age, familial history, height, and weight, BMI, and METs at week 0 and week 8 were not alteration between the two groups (Table 1).

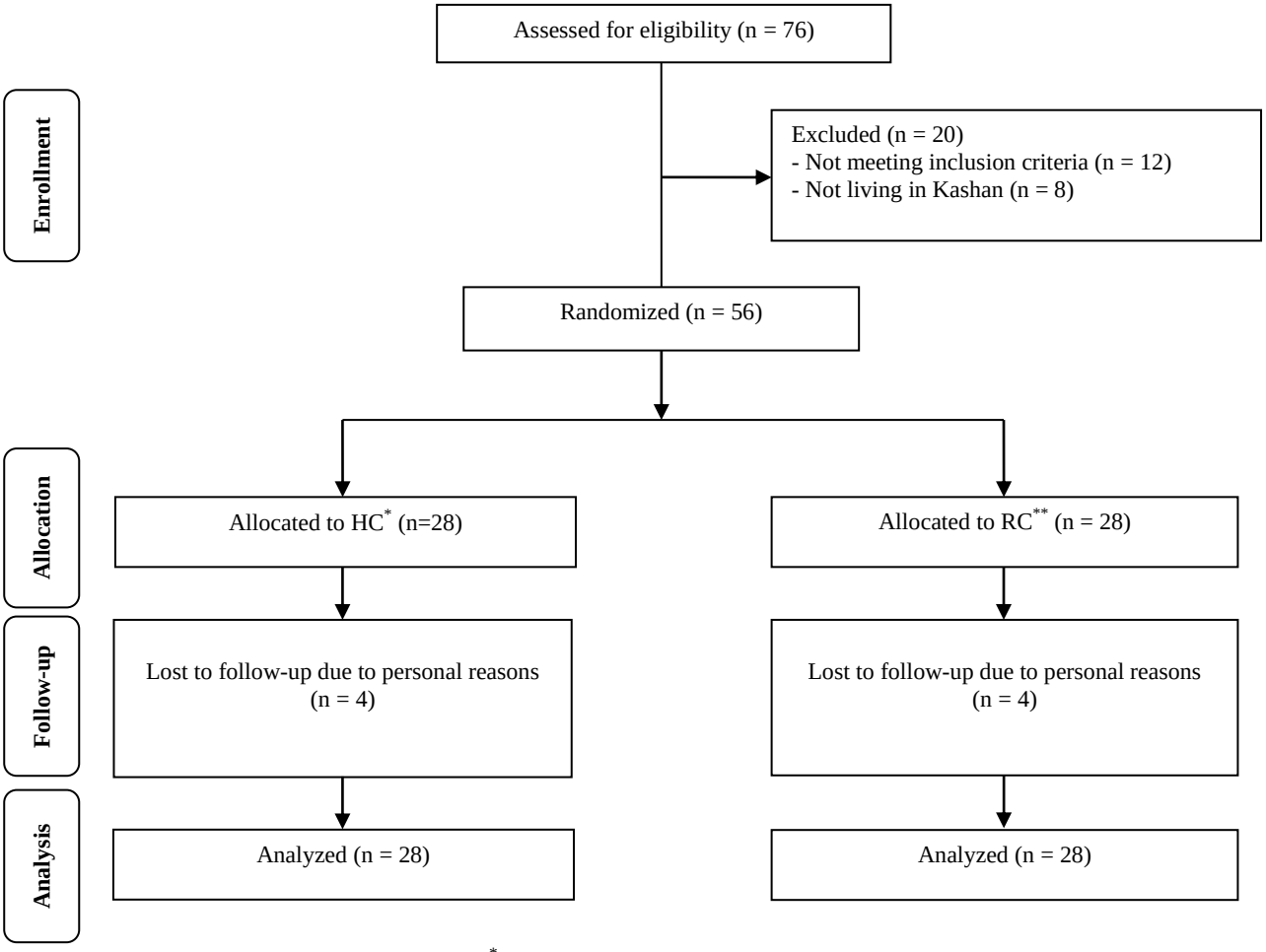

Figure 1. Summary of patient flow diagram. * High-carbohydrate diet: Energy-restricted diet that contained 60-65% of energy from carbohydrates, 20-25% from fats, and 14-18% from proteins. ** Restricted carbohydrate diet: Energy-restricted diet that contained 43-49% of energy from carbohydrates, 36-40% from fats, and 14-18% from proteins
HC: High-carbohydrate; RC: Restricted carbohydrate

Based on the 3-day records, no significant alteration was seen between the two groups in terms of macro- and micronutrients (Table 2).

RC diet decreased FPG (−11.5 ± 28.3 vs. +7.0 ± 26.9 mg/dl, P = 0.010) and hs-CRP (−564.3 ± 1280.1 vs. +286.1 ± 1789.2 ng/ml, P = 0.040) compared with a HC diet (Table 3). In addition, compared with a HC diet, RC diet increased TAC (+274.8 ± 111.5 vs. +20.2 ± 82.5 mmol/l, P < 0.001) and GSH levels (+51.6 ± 111.5 vs. −32.6 ± 88.5 µmol/l, P = 0.003). Within-group changes revealed a significant decrease of FPG (P = 0.040), serum hs-CRP (P = 0.020), and a significant increase of plasma TAC (P < 0.001), and GSH levels (P = 0.020) in the RC diet.

When we controlled the analysis for baseline values of biochemical indicators, age, BMI at week 0, METs change and familial history, findings unaltered (Table 4).

Discussion

We illustrated that RC diet for 8 weeks among overweight diabetic persons with CHD had useful effects on FPG, hs-CRP, plasma TAC, and GSH values; however, it did not influence other metabolic profiles. We did not randomize participants based on their plasma MDA levels because all participants were T2DM and CHD. Random assignment to two groups was done after stratification according to age, BMI, gender, and the dosage and kind of medications and random assignment were done by the use of computer-generated random numbers.

T2DM patients are susceptible to metabolic complications.[26,27] We found that RC diet compared with HC diet in overweight persons with T2DM and CHD for 8 weeks decreased FPG levels, but unaltered insulin metabolism and lipid profiles.

Table 1. General characteristics of study participants

Characteristics	HC diet* (n = 28)	RC diet** (n = 28)	P***
Familial history (%)	10 (35.7)	10 (35.7)	> 0.999†
Smoking (%)	2 (7.1)	2 (7.1)	> 0.999†
Aspirin 80 mg (%)	28 (100)	28 (100)	> 0.999†
Statin (%)	28 (100)	28 (100)	> 0.999†
Insulin therapy (%)	6 (21.4)	5 (17.9)	0.730†
Antidiabetic drugs (%)			
Monotherpy	16 (72.7)	16 (69.6)	
Combination therapy	6 (27.3)	7 (30.4)	0.810†
Hypertension (%)	19 (67.9)	20 (71.4)	0.770†
ACEI/ARB drugs (%)	28 (100)	28 (100)	> 0.999†
Blocker drugs (%)			
β-blocker	26 (92.9)	27 (96.4)	
Calcium channel blocker	2 (7.1)	1 (3.6)	0.550†
Duration of DM (year)	6.3 ± 5.0	6.6 ± 4.8	0.850
Duration of CHD (year)	8.2 ± 4.7	8.8 ± 4.0	0.620
HbA1c (mmol/mol)	56.5 ± 4.7	54.9 ± 6.9	0.300
SBP at study baseline (mmHg)	135.7 ± 7.9	133.2 ± 13.3	0.370
SBP at end-of-trial (mmHg)	136.2 ± 8.0	133.5 ± 13.8	0.360
SBP change (mmHg)	0.5 ± 1.4	0.3 ± 1.1	0.680
DBP at study baseline (mmHg)	84.1 ± 8.4	85.2 ± 8.0	0.620
DBP at end-of-trial (mmHg)	84.3 ± 8.5	85.1 ± 7.8	0.750
DBP change (mmHg)	0.2 ± 1.2	−0.1 ± 1.1	0.200
Age (year)	65.2 ± 11.6	61.1 ± 9.9	0.1500
MET-h/day change	−0.3 ± 0.8	−0.3 ± 0.9	0.850
Height (cm)	156.8 ± 15.9	157.6 ± 11.2	0.820
Weight at study baseline	79.0 ± 14.8	77.6 ± 11.7	0.690
Weight at end-of-trial	76.8 ± 14.3	75.5 ± 11.9	0.700
Weight change (kg)	−2.2 ± 2.1	−2.1 ± 1.7	0.880
BMI at study baseline	32.2 ± 4.4	31.2 ± 3.5	0.360
BMI at end-of-trial	31.3 ± 4.4	30.4 ± 3.6	0.360
BMI change (kg/m^2)	−0.9 ± 0.8	−0.9 ± 0.7	0.960
MET-h/day at study baseline	26.4 ± 2.0	27.0 ± 1.7	0.210
MET-h/day at end-of-trial	26.1 ± 2.1	26.7 ± 1.7	0.250

Data are means ± SDs. * HC diet: Energy-restricted diet that contained 60-65% of energy from carbohydrates, 20-25% from fats, and 14-18% from proteins. ** RC diet: Energy-restricted diet that contained 43-49% of energy from carbohydrates, 36-40% from fats, and 14-18% from proteins. *** Obtained from independent samples student's t-test. † Obtained from Pearson chi-square test. BMI: Body mass index; ACEI: Angiontensin converting enzymes inhibitors; ARB: Aldosterone receptor blockers; CHD: Coronary heart disease; DBP: Diastolic blood pressure; METs: Metabolic equivalents; HbA1c: Hemoglobin A1c; DM: Diabetes mellitus; SBP: Systolic blood pressure; SD: Standard deviation; HC: High-carbohydrate; RC: Restricted carbohydrate

In a study by Ballard et al.[28] was seen that carbohydrate limited diets for 6 weeks decreased insulin, HOMA-IR and triglycerides values, but unchanged FPG and other lipid fractions. A meta-analysis study has shown that changes in values of hemoglobin A1c, FPG, total- and low-density lipoprotein-cholesterol did not differ significantly between the HC and low-carbohydrate groups.[12] However, the HC diet significantly increased insulin and triglycerides concentrations by 8% and 13%, respectively, and lowered HDL-cholesterol by 6% compared with the low-carbohydrate diet.[12] A significant improvement of lipid profiles was seen following the consumption of a carbohydrate-restricted diet among patients with MetS.[29]

This study demonstrated that compared with an HC diet, adherence to RC diet for 8 weeks decreased serum hs-CRP and increased plasma TAC and GSH concentrations, while it did not affect plasma NO and MDA in overweight diabetic patients with CHD. In line with our study, in a parallel randomized clinical trial, Forsythe et al.[30] demonstrated that a very-low-carbohydrate diet (12% of energy from carbohydrate) led to a greater reduction in some inflammatory markers compared to a low-fat diet among overweight men and women with atherogenic dyslipidemia. In addition, in another study among insulin-resistant patients, moderate carbohydrate restriction for 12 weeks resulted in lower inflammatory marker concentrations compared to fat restriction.[31]

Table 2. Dietary intakes of study participants throughout the study

Dietary intakes	HC diet* (n = 28)	RC diet** (n = 28)	P***
Energy (kcal/day)	1679.0 ± 78.0	1652.0 ± 72.0	0.200
Carbohydrates (g/day)	265.7 ± 16.9	200.0 ± 9.2	< 0.001
MUFAs (g/day)	15.9 ± 0.1	26.7 ± 1.3	< 0.001
Fat (g/day)	46.5 (46.5, 46.9)	73.0 (70.1, 74.6)	< 0.001†
SFAs (g/day)	16.6 (16.6, 16.9)	23.1 (22.0, 23.5)	< 0.001†
PUFAs (g/day)	9.8 (9.7, 9.8)	18.6 (17.2, 18.7)	< 0.001†
Protein (g/day)	57.1 (57.1, 62.1)	60.9 (57.2, 62.0)	0.100†
Cholesterol (mg/day)	111.3 (0.8)	133.2 (8.5)	< 0.001†

Values are means ± SDs for normally distributed variables and median (Q1,Q3) for non-normally distributed variables. * HC diet: Energy-restricted diet that contained 60-65% of energy from carbohydrates, 20-25% from fats, and 14-18% from proteins. ** RC diet: Energy-restricted diet that contained 43-49% of energy from carbohydrates, 36-40% from fats, and 14-18% from proteins. *** Obtained from independent samples student's t-test. † Obtained from Mann-Whitney test. MUFAs: Monounsaturated fatty acids; PUFAs: Polyunsaturated fatty acids; SFAs: Saturated fatty acids; SD: Standard deviation; HC: High-carbohydrate; RC: Restricted carbohydrate

Barbosa et al.[32] also indicated that low energy and carbohydrate intake for 2 months were associated with higher TAC levels in apparently healthy adults. Moreover, the energy intake limitation by 2000 kJ among obese persons decreased oxidative stress.[33]

Table 3. Effect of a restricted carbohydrate (RC) diet on metabolic profiles, biomarkers of inflammation and oxidative stress at baseline and 8 weeks after the intervention in patients with overweight type 2 diabetes mellitus (T2DM) and coronary heart disease (CHD)

Variables	HC diet* (n = 28)				RC diet** (n = 28)				P†
	Baseline	End-of-trial	Change	P***	Baseline	End-of-trial	Change	P***	
FPG (mg/dl)	124.1 ± 41.9	131.1 ± 48.9	7.0 ± 26.9	0.170	134.3 ± 54.9	122.7 ± 52.6	−11.5 ± 28.3	0.040	0.010
Insulin (µIU/ml)	15.1 ± 7.3	13.9 ± 5.9	−1.2 ± 4.5	0.160	12.5 ± 4.8	11.3 ± 4.9	−1.2 ± 3.0	0.050	0.930
HOMA-IR	4.5 ± 2.1	4.2 ± 2.1	−0.3 ± 1.2	0.180	4.1 ± 2.0	3.7 ± 2.0	−0.4 ± 1.0	0.050	0.800
HOMA-B	45.1 ± 30.0	40.9 ± 24.1	−4.2 ± 16.4	0.180	33.8 ± 17.9	30.4 ± 16.9	−3.4 ± 10.1	0.080	0.820
QUICKI	0.31 ± 0.02	0.31 ± 0.02	0.001 ± 0.01	0.370	0.31 ± 0.02	0.32 ± 0.02	0.01 ± 0.01	0.120	0.430
Triglycerides (mg/dl)	130.5 ± 42.6	144.7 ± 82.7	14.2 ± 81.3	0.360	119.8 ± 45.5	126.5 ± 44.0	6.7 ± 31.8	0.270	0.650
VLDL−cholesterol (mg/dl)	26.1 ± 8.5	28.9 ± 16.5	2.8 ± 16.2	0.360	24.0 ± 9.1	25.3 ± 8.8	1.3 ± 9.3	0.270	0.650
Total cholesterol (mg/dl)	154.7 ± 32.4	155.1 ± 42.9	0.5 ± 28.8	0.920	145.1 ± 30.7	148.1 ± 30.8	3.0 ± 35.0	0.650	0.760
LDL-cholesterol (mg/dl)	77.5 ± 26.7	76.2 ± 36.4	−1.2 ± 27.0	0.810	83.4 ± 28.1	84.0 ± 25.4	0.7 ± 31.2	0.910	0.810
HDL-cholesterol (mg/dl)	51.0 ± 10.0	49.9 ± 9.3	−1.1 ± 7.6	0.440	37.8 ± 7.5	38.8 ± 7.0	1.0 ± 7.2	0.450	0.280
Total-/HDL-cholesterol	3.1 ± 0.8	3.1 ± 0.7	0.0 ± 0.7	0.760	3.9 ± 1.0	3.9 ± 0.8	0.0 ± 0.8	0.640	0.580
hs-CRP (ng/ml)	2348.0 ± 1925.3	2634.1 ± 1897.0	286.1 ± 1789.2	0.400	2346.4 ± 1730.7	1782.1 ± 1254.5	−564.3 ± 1280.1	0.020	0.040
NO (µmol/l)	42.1 ± 9.9	45.0 ± 8.8	2.9 ± 9.2	0.100	55.7 ± 6.0	57.0 ± 7.3	1.3 ± 4.8	0.150	0.420
TAC (mmol/l)	862.0 ± 169.4	882.2 ± 178.4	20.2 ± 82.5	0.200	943.2 ± 154.3	1218.0 ± 160.9	274.8 ± 111.5	< 0.001	< 0.001
GSH (µmol/l)	403.5 ± 100.6	370.9 ± 57.8	−32.6 ± 88.5	0.060	419.0 ± 105.5	470.6 ± 90.9	51.6 ± 111.5	0.020	0.003
MDA (µmol/l)	3.1 ± 0.7	3.1 ± 0.5	0.0 ± 0.7	0.920	2.5 ± 0.6	2.7 ± 0.4	0.2 ± 0.4	0.020	0.240

All values are means ± SDs. * HC diet: Energy-restricted diet that contained 60-65% of energy from carbohydrates, 20-25% from fats, and 14-18% from proteins. ** RC diet: Energy-restricted diet that contained 43-49% of energy from carbohydrates, 36-40% from fats, and 14-18% from proteins. *** P values represent paired-samples t-test. † P values represent independent samples student's t-test. CHD: Coronary heart disease; FPG: Fasting plasma glucose; GSH: Total glutathione; HOMA-IR: Homeostasis model of assessment-estimated insulin resistance; HOMA-B: Homeostasis model of assessment-estimated B cell function; hs-CRP: High-sensitivity C-reactive protein; RC: Moderately restricted carbohydrate; MDA: Malondialdehyde; NO: Nitric oxide; QUICKI: Quantitative insulin sensitivity check index; TAC: Total antioxidant capacity; T2DM: Type 2 diabetes mellitus; SD: Standard deviation; HC: High-carbohydrate; RC: Restricted carbohydrate; VLDL: Very-low-density lipoprotein; LDL: Low-density lipoprotein; HDL: High-density lipoprotein

Table 4. Adjusted changes in metabolic variables in patients with overweight type 2 diabetes mellitus (T2DM) and coronary heart disease (CHD)

Variables	HC diet* (n = 28)	RC diet** (n = 28)	P***
FPG (mg/dl)	6.900 ± 5.100	−11.300 ± 5.100	0.010
Insulin (µIU/ml)	−0.600 ± 0.700	−1.700 ± 0.700	0.250
HOMA-IR	−0.200 ± 0.200	−0.500 ± 0.200	0.350
HOMA-B	−2.100 ± 2.200	−5.500 ± 2.200	0.310
QUICKI	0.004 ± 0.002	0.006 ± 0.002	0.060
Triglycerides (mg/dl)	14.000 ± 11.400	6.900 ± 11.400	0.660
VLDL-cholesterol (mg/dl)	2.800 ± 2.300	1.400 ± 2.300	0.660
Total cholesterol (mg/dl)	2.800 ± 6.000	0.700 ± 6.000	0.800
LDL-cholesterol (mg/dl)	−1.300 ± 5.200	0.700 ± 5.200	0.770
HDL-cholesterol (mg/dl)	1.600 ± 1.400	−1.700 ± 1.400	0.150
Total-/HDL-cholesterol ratio	−0.100 ± 0.100	0.100 ± 0.100	0.210
hs-CRP (ng/ml)	232.100 ± 260.000	−514.300 ± 260.000	0.040
NO (µmol/l)	0.300 ± 1.500	3.900 ± 1.500	0.140
TAC (mmol/l)	9.400 ± 18.100	285.600 ± 18.100	< 0.001
GSH (µmol/l)	−41.300 ± 13.400	60.300 ± 13.400	< 0.001
MDA (µmol/l)	0.200 ± 0.100	0.003 ± 0.100	0.130

All values are means ± standard error. Values are adjusted for baseline values, age, BMI at baseline, METs change and familial history. * HC diet: Energy-restricted diet that contained 60-65% of energy from carbohydrates, 20-25% from fats, and 14-18% from proteins. ** RC diet: Energy-restricted diet that contained 43-49% of energy from carbohydrates, 36-40% from fats, and 14-18% from proteins. *** Obtained from ANCOVA. FPG: Fasting plasma glucose; GSH: Total glutathione; HOMA-IR: Homeostasis model of assessment-estimated insulin resistance; HOMA-B: Homeostasis model of assessment- estimated B-cell function; hs-CRP: High-sensitivity C-reactive protein; MDA: Malondialdehyde; METs: Metabolic equivalents; NO: Nitric oxide; QUICKI: Quantitative insulin sensitivity check index; TAC: Total antioxidant capacity; HC: High-carbohydrate; VLDL: Very-low-density lipoprotein; LDL: Low-density lipoprotein; HDL: High-density lipoprotein

However, consumption of an RC diet compared with an HC diet did not affect any significant effect on inflammatory biomarkers among women with MetS for 6 weeks.[15] In another study by Rankin and Turpyn.,[34] low carbohydrate diet compared with HC diet increased CRP during weight loss among overweight women for 4 weeks. The previous studies have shown that acute ingestion of carbohydrate clearly induces ROS, inflammation and oxidative stress.[13,14] Decreased total saturated fatty acids, palmitoleic acid levels, down-regulation of nuclear factor-kappa B and cyclooxygenase-2 expression following the consumption of an RC diet may result in its anti-inflammatory and anti-oxidative effects.[30,35] It must be considered that consumption of the MRC diet significantly decreased serum hs-CRP (564.3 ng/ml), and increased plasma TAC (274.8 mmol/l), and GSH (51.6 µmol/l) concentrations in this study.

One strength of our study was an assessment of metabolic status and its randomized design. One of our limitations was no examine the compliance to the RC and HC eating plan. In addition, the current 8-week diet intervention in a group of among 56 overweight diabetic patients with CHD could be viewed as short in duration and small in sample size in comparison to larger clinical trials. In our study, persons were overweight patients with T2D and CHD aged 40-85 years old. We believe that adherence to the same diets in different age ranges may have different outcomes. Therefore, this should be taken into account in the explanation of our findings.

Conclusion

RC diet for 8 weeks among overweight diabetic patients with CHD had useful effects on some metabolic indices. This offers an RC diet with high unsaturated and low saturated fat may allow the advantageous remedial potential for overweight persons with T2DM and CHD handling.

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Conflict of Interests

Authors have no conflict of interests.

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