

Correlation between Dietary Glycemic Index and Blood Lipids Abnormality as a Main Risk Factor of Atherosclerosis in Healthy Women from Ahvaz

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Abstract

Background: Atherosclerosis and subsequent coronary artery disease is the leading cause of death in Iranian population. High serum lipid levels, especially the elevated level of low-density lipoprotein (LDL), have been shown to be strongly related to the development of atherosclerosis. The relationship between dietary glycemic index (GI) and lipid profile, particularly in non-western populations, has not been well studied; also, the result of studies are inconsistent. The aim of this study was to evaluate the relationship between dietary glycemic index (GI) and main risk factor of atherosclerosis including abnormal blood lipid levels in healthy women.

Methods: This cross-sectional study was done to investigate the associations between dietary GI and lipid profile. The subjects were 87 female personnel of Ahvaz Jundishapur University of Medical Sciences aged 25-55 y; they were recruited randomly. Dietary GI was calculated from six 24 hour recalls (including 4 usual days and 2 holidays).

Results: The mean of dietary GI was 72.1 ± 4.07 . After adjustment for potential dietary and non-dietary confounding factors, no significant relationship was found between dietary GI with HDL-C and LDL-C. There was also no statistically significant relationship between GI and total cholesterol or fasting Triacylglycerol.

Conclusion: Findings of this study did not support the hypothesis of physiologic relevance of GI and lipid profile abnormality as a potential risk factor for atherosclerosis.

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Introduction

Globally, cardiovascular disease is the main cause of death. In 2005, cardiovascular disease accounted for approximately thirty percent of deaths worldwide. Also, CHD is a leading cause of mortality, morbidity, and disability in Iranian population. It accounts for nearly 50 percent of all deaths per year.¹

A significant number of Iranians have one or more

major risk factors for CHD. Cardiovascular disease is one of the major health and social problems. Although many risk factors have been suggested for CHD in Iranian population, we focused on atherosclerosis and its components including abnormal lipid levels.

Lifestyle patterns including diet may be related to atherosclerosis.² Recent studies focused on dietary carbohydrate, an agent increasing serum LDL-C total cholesterol and TG and decreasing HDL-C.³

Carbohydrates vary according to their ability to increase postprandial blood glucose levels. This can be described as glycemic index (GI). The GI concept was introduced by Jenkins and colleagues, early in 1980's.³ The glycemic index concept is an extension of the fiber hypothesis, suggesting that fiber consumption reduces the rate of nutrient influx from the gut. The glycemic index has a particular relationship to those chronic western diseases associated with abdominal obesity and insulin resistance. Early studies indicated that starchy carbohydrate foods have a very different effect on postprandial blood glucose and insulin response in healthy and diabetic subjects, depending on the rate of digestion. A range of factors associated with food consumption was later shown to alter the rate of glucose absorption and subsequent glycemia and insulinemia. At this stage, systemic documentation of the differences existing among carbohydrate foods provided a numeric physiologic classification of relevant carbohydrate foods in the prevention and treatment of diseases such as diabetes. Then, low glycemic index diet has been shown to lower the urinary c-peptide excretion in healthy subjects, improve glycemic control in diabetic subjects, and reduce serum lipids in hyperlipidemic subjects. Furthermore, consumption of low glycemic index diets have been associated with higher HDL-cholesterol concentration, and, in large cohort studies, with decreased risk of developing diabetes and cardiovascular disease.^{4,5} The glycemic index is an indicator of how quickly dietary carbohydrate increases the blood glucose compared with a reference food.^{4,5} GI is a measure of how much each carbohydrate-containing food raises the blood glucose compared with a standard food of either glucose or white bread (per 50g available carbohydrate).³

GI was originally designed to help people with diabetes while selecting food.³ It seems that dietary GI has increased at global level recently. It is partly because of increased carbohydrate intake and of changing of food processing.⁶ Diets with higher GI are positively associated with CHD,⁷ probably because they have adverse effects on serum lipid and glucose levels.

Objective

Because of limited number of studies in Asian countries,⁸⁻¹⁰ the present study was carried out to further investigate the relationship between dietary GI and blood lipid levels.

Patients and Methods

A cross-sectional study was conducted to explore the associations between dietary GI and lipid profile. The subjects were 87 healthy female aged 25-55 years. The

sample size was calculated according to the following formula.

$$\ln [(1+r)/(1-r)] = C \times 0.5$$

$$\ln [(1+0.36)/(1-0.36)] = 0.37 \times 0.5$$

$$C = 0.37$$

$$\alpha = 0.05$$

$$n = [(z_{\alpha/2} + z_{1-\beta}) \div C] + 3$$

$$B = 0.1 \quad n = [(1.96 + 1.25) \div 0.37] + 3 = 82$$

100 people were considered as sample and only 87 of them remained until the end of the trial. The subjects were personnel of Ahvaz Jundishapur University of Medical Sciences, who were recruited randomly. After taking informed written consent, the subjects underwent a brief physical examination, including height and weight. The approval number of the ethics committee for the study in Ahvaz University was 15/A.K. This study was conducted in 2010.

The exclusion criteria were previously diagnosed diabetes or cardiovascular disease, intake of extremely high or low calorie intake, (<600 or >4000 kcal/d) and endocrine diseases such as hypo- or hyperthyroidism and the women who were not pregnant or were not in the lactation period. No subject took blood lipid lowering medication or any drug that affected the lipid profile.

Weight was measured after an overnight fasting and with minimum clothing. Height was measured according to a standard method. Body mass index (BMI) was calculated as weight (kg) divided by the height squared (m). A morning venous blood sample was taken after 12 hours fasting. Blood was collected in evacuated tubes, and the serum was separated by centrifugation of the blood at 3000 \times g for 10 min at room temperature. Then, HDL-C, triacylglycerol, and T-chol were measured by enzymatic assay methods. Serum LDL-C concentration was calculated by using Fried Wald equation.¹¹

Dietary Assessment

Six 24 hour recalls were collected by a trained dietitian in order to estimate the nutritional intake (4 usual days and 2 holidays). Before joining the subject to the research, a dietitian instructed each subject on how to report the detailed description of all foods consumed, ingredients, cooking methods and also portion sizes. Data on the frequency and duration of exercise, exercise time (minute /week), and history of illnesses were collected by a questionnaire.

Calculation of Dietary GI

A table of white bread-based GI food list containing Iranian foods¹² and foods listed in international GI

table¹³ was used for the calculation of dietary GI. Firstly, the food items were listed (six 24 hour food recalls of all subjects contained 351 different foods, among which 109 items (29%) were foods containing more than 3g carbohydrate per serving). Secondly, food items without carbohydrate were excluded and GI of carbohydrate containing foods was extracted from Iranian table of GI. The next step involved matching the foods that did not exist in Iranian table directly to those in the international table of GI. Carbohydrate content of the food (g/serving) was extracted by Nut 4 software. The dietary GI was determined by multiplying the amount of carbohydrate content (g) of the food item by the food GI. The sum of these products was then divided by the total daily carbohydrate intake.¹⁴

$$\text{Dietary GI} = \sum \frac{\{(GI \text{ of food item}) \times (\text{grams of carbohydrate of food item})\}}{\text{Total daily carbohydrate intake}}$$

The results were reported as mean±SD. To confirm the correlation between dietary GI and lipid profile, the subjects were divided into groups of GI, according to literature.¹² The cut-off points of GI were as follows: low GI (55-60), medium GI (60-69) and high GI (>70), but because of low number of GI lower than 60, the first two groups were combined together, so GI was put in two groups: GI lower than 70, and GI upper than 70.

We examined the correlation between dietary GI and lipid profile using Pearson's correlation coefficients between dietary GI and serum level of HDL-c, LDL-c, T chol and TG. Also, to confirm the correlation between dietary GI and lipid profile, the subjects were divided into two groups of dietary GI. The differences between these two groups were analyzed by independent sample t-test. The differences were considered statistically significant if P value was <0.05. All statistical analyses were performed using SPSS software, version 17.

Results

The subjects were 87 women aged 23 to 55 years old with a mean age of 36±7.7 years and a mean BMI of 25.6±4.52 kg/m². The mean daily energy intake and mean dietary GI were 1507±491 Kcal and 72.1±4.07, respectively. Moreover, the mean intake of carbohydrate was 211.5±57 gr.

Table 1 shows that there isn't any difference between lipid profile and BMI between two groups of participants based on dietary GI intake.

As shown in Table 2, there wasn't any correlation between dietary GI and lipid profile of the women participating in this study.

By considering factors such as age, physical activity time (Table 3) and calorie intake per day and controlling them, dietary GI was not associated with HDL-c (P=0.70, r=0.343), LDL_C (P=0.073, r=0.137), TC (P=0.68, r=0.261) and TG (P=0.91, r=0.208). (Tables 1 and 2)

There were no significant differences in age between the two groups of GI. Also, dietary GI was not significantly different in 3 levels of physical activity (Table 3).

Table 2: Correlation between dietary glycemic index and lipid profile

Lipid profile	Correlation coefficient
HDL-c	0.343
LDL-c	0.137
TC	0.261
TG	0.208

Table 3: Comparison of the average dietary GI in terms of physical activity

Physical activity	Glycemic index(GI) Mean±SD
Very low	72.45±3.88
Low	69.4±5.39
Moderate	72.6±3.12
P=0.084	

Discussion

Because limited number of studies have been published in Asian countries such as Iran (only one),¹⁰ we examined the correlation between dietary GI and serum lipid levels among 87 healthy women in Ahvaz city. We found that dietary GI was not associated with T-chol, triacylglycerol, LDL-C, HDL-C. Different studies have reported different results, and several factors have been suggested for these different results.

In a cohort study (from 2001 to 2008) by Mirmiran and colleagues, that was done on 120 individual older

Table 1: Adjusted mean (95% CI) of lipid profile and BMI according to 2 groups of glycemic index among the participants

MEAN SCORE	GI<70	GI>70	P*value
TC	193.4±41.25	189.9±31.35	0.68
HDL-C	46.3±10.07	45.6±7.37	0.7
LDL-C	127.09±32.1	124.7±26.1	0.073
TG	99.4±43.6	98.1±51.4	0.91
BMI	3.74±25.6	4.68±25.3	0.78

*Independent sample t-test

than 40 years of age from Tehran and without diabetes or metabolic syndrome individuals on higher quintile of GI had increased the levels of T-chol, LDL-C and decreased level of HDL-C. In addition, the higher quintile of GI accompanied higher BMI of individuals.¹⁰

Both dietary GI and GL were positively correlated with fasting triacylglycerol in 2 cross-sectional studies;^{8,15} however, no association between dietary GI and fasting triacylglycerol was observed in a study of the elderly men.¹⁵ Regarding the correlation between dietary GI and HDL-C, most of studies^{8,17,18} but not all^{15,16} have shown a positively inverse relationship.

Some studies have shown that there was no relationship between dietary GI, LDL-C and total cholesterol and this result is similar to our findings.^{9,12,16}

In addition to these studies, some clinical trials have been done showing different results. Of the two cross-over studies that reported total, LDL and HDL cholesterol concentrations, contradictory results were reported, one showing a significant increase¹⁹ and the other a significant decrease with the low GI compared to high GI diet for all three variables.²⁰ Also, the diet type had no significant effect on the TG concentrations in the two studies reporting these data.^{19,20}

Parallel studies have shown no significant difference in total, LDL, HDL and very low density lipoprotein (VLDL) cholesterol, and TG concentrations between low GI and high GI diet.²¹⁻²³

Proposed mechanism that can explain the correlation between dietary GI and blood lipid levels is as follows: there is strong evidence that diets with high GI may directly increase insulin resistance through their effects on glycemia, free fatty acids, and counter regulatory hormone secretion. Insulin resistance seems to cause an increase in triglycerides and a decrease in HDL-C.^{6,24,25} Also, we found that dietary GI did not correlate with BMI.

Different studies have reported different results, and several factors have been suggested for these different results. Although our results are similar to some studies,^{9,12,16} opposite results can be explained in this way. As the validity of self-reported energy and nutrient intake is the key to the interpretation of many epidemiological studies, so different results in our study may be contributed to the possibility of underestimation and overestimation of dietary intake in all self-reported dietary assessment. Also, underreporting of the subjects may play a role that could be corrected by excluding the under reporters. Because of limited sample size in our study, the exclusion of under reporters was not feasible.

Conclusion

Our findings suggest that the quality of carbohydrate

consumption is not related to blood lipid levels as risk factors of atherosclerosis in healthy subjects. Our data do not support the hypothesis that diets with a low GI are related to favorable lipid profile and lower BMI.

Conflict of Interest: None declared.

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