Original Communication

Serum Antioxidant Vitamin Levels in Patients with Coronary Heart Disease

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Abstract: The aim of this study was to investigate anthropometric measurements, body composition, and serum antioxidant vitamin levels in men with coronary heart disease (CHD). Thirty-five men with CHD and 31 men without CHD, aged 40–65 years, were included this study. Dietary records and anthropometric measurements of each participant were recorded by researchers and serum antioxidant vitamin levels and lipid profiles were analyzed. Fat mass (FM) and the percentage of fat mass (FM%) in men with CHD was higher than in men without CHD ($p < 0.05$). Lipid profiles were found to be similar in both groups, with the exception of high-density lipoprotein cholesterol (HDL-C). Men with CHD had lower HDL-C levels than men without CHD ($p < 0.05$). When the antioxidant vitamin intake of participants was investigated, vitamin E intake in men without CHD was found to be less than in men with CHD ($p < 0.05$). However, serum vitamin A, vitamin E, and vitamin C levels in men with CHD were found to be lower than in men without CHD ($p < 0.05$). Based on the results of this study, we propose that high FM, low HDL-C, and low serum antioxidant vitamin levels could be important risk factors for CHD.

Keywords: Coronary heart disease, antioxidants, vitamin A, vitamin E, vitamin C, body composition, lipid profile

Introduction

Complications from Coronary Heart Disease (CHD) depend on the degree of atherosclerosis, and often include myocardial ischemia and necrosis [1]. Coronary heart disease is one of the leading causes of death in the United States and many industrialized countries [2]. In Turkey, even though the general population is relatively younger, the prevalence of CHD is unexpectedly high. In addition, the mortality rate from CHD in Turkey is 5 % per year, higher than in many European countries [3].

Atherosclerosis is the main cause of CHD [4] and lipid peroxidation plays a central role in the atherosclerotic process. When polyunsaturated fatty acids in cell membranes are exposed to oxidation, peroxyl radicals are formed, leading to chain reactions [5]. Excessive free radical intake from the environment, or increasing amounts of free radical byproducts of metabolic activities, increases low-density lipoprotein (LDL) oxidation. Oxidized LDL then causes oxidative changes by attacking the extracellular matrix of the arterial wall with scavenger receptors, forming a basis for fatty plaques. Under normal conditions, antioxidant systems protect the organism from oxidative damage and oxidation of lipoproteins can be blocked by antioxidants [6, 7].

To date, most epidemiological studies have shown that high serum levels of antioxidant vitamins, such as vitamin E, vitamin C, and vitamin A play important roles in the prevention of CHD [5, 6, 8, 9]. With this background, the present study was performed to evaluate the antioxidant effects of vitamin A, vitamin C, and vitamin E by investigating dietary intake and serum levels of these vitamins in men with CHD.

Subjects and methods

The present study was conducted on men between 40–65 years of age and included 35 men with CHD and 31 men without CHD. The men in the CHD group were recently diagnosed with CHD and had no other chronic diseases. CHD diagnoses were confirmed by coronary angiography, re-vascularization, a history of positive treadmill exercise tests, thallium scintigraphy, detection of perfusion defects, a history of Q-wave or Q-wave myocardial infarction, and stable or unstable angina. All diagnoses were conducted at the Cardiology Department of Hacettepe University Hospital, and patients had not yet received treatment before the study period. No individuals in the study group were using vitamin-mineral supplements.

A questionnaire was filled out by researchers based on interviews with study participants to collect general information. Dietary records from three consecutive days (one day of which was on a weekend) were collected to determine antioxidant vitamin intake levels, and these records were evaluated using the Nutrition Information System (BeBiS) program [10].

Weight measurements were recorded in the morning following overnight fasting (12 hours), with study participants wearing light clothes and no shoes. A portable scale was used to measure body weight to the nearest half-kilogram. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer, with feet together and the head in the Frankfort plane [11]. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Body composition measurements were estimated using the bio-electrical impedance (BIA) method with Body Stad 1500. Each study participant agreed to adhere to the conditions necessary for BIA measurements including: no heavy physical activity for 24–48 hours, no alcohol for 24 hours, no eating or drinking for at least 2–4 hours, and no tea, coffee, cola, or smoking for at least 4 hours before the BIA measurements [12].

Early-morning venous blood samples were obtained from each study participant for biochemical screening tests following a 12-hour, overnight fast. Lipid profiles, including total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein-cholesterol (VLDL-C), and triglycerides (TG) were analyzed. Professional staff performed venipuncture using vacutainers to obtain 15 mL of whole blood. Blood was centrifuged for plasma separation at Hacettepe University Hospital, where the actual biochemical analyses were performed. Roche Diagnostic Kits were used for TG, HDL-C, and TC analysis. LDL-C was calculated using the formula reported by Friedewald *et al.:* LDL-C = TC – (HDL-C + (TG/5)) [13]. Serum concentrations of vitamin A and vitamin E were measured by high-performance liquid chromatography (HPLC) (ClinRep® Complete Kit for Vitamin A, E, and C determination in Plasma, Recipe, Germany) at the Ibni-Sina Hospital Biochemistry Laboratory.

This study was found to be medically appropriate by the Hacettepe University Medical Faculty of Medicine, Surgery and Medicine Research Ethics Board (Approval Number: FON 04/4–11) and each participant signed a consent form.

All values are reported as the mean ($\bar{x}$) ± standard deviation (SD). Data were analyzed using the Statistical Package for Social Sciences Software for Windows 13.0 (SPSS Inc. Chicago, IL, USA). The Kolmogorov-Smirnov test was used to determine whether outcome variables were normally distributed. To compare normally distributed data, a *t*-test was used. The Mann-Whitney *U*-test was used for non-normally distributed data. P-values less than 0.05 were considered to be statistically significant for all analyses.

Results

Fat mass (FM), fat mass percentage (FM%), and fat-free mass percentage (FFM%) were found to be statistically different between the two study groups

($p < 0.05$). Specifically, men with CHD had higher FM and FM% and lower FFM% than men without CHD (Table I).

Table II shows a comparison of serum lipid profiles from men with CHD and men without CHD. Serum TC, TG, LDL-C, VLDL-C, and TC/HDL-C ratios were found to be similar between the groups ($p > 0.05$). However, serum HDL-C levels were found to be higher in men without CHD (49.8 ± 11.4 mg/dL) than in men with CHD (42.5 ± 12.3 mg/dL) ($p < 0.05$)

Antioxidant vitamin intake levels from three-day food consumption records are shown in Table III. Vitamin E intake levels in men without CHD were lower than in men with CHD ($p < 0.05$), while vitamin A and C intake levels in both groups were similar ($p > 0.05$). However, serum antioxidant vitamin levels between the two groups were different, and men without CHD had higher serum vitamin A, E, and C levels than men with CHD ($p < 0.05$). In men without CHD, mean serum antioxidant vitamin levels were: serum vitamin A, 0.8 ± 0.4 mg/L; serum vitamin E, 17.2 ± 7.1 mg/L; and serum vitamin C, 34.7 ± 9.2 μmol/L. In contrast, mean serum antioxidant vitamin levels in men with CHD were measured to be: serum vitamin A, 0.6 ± 0.2 mg/L; serum vitamin E, 14.0 ± 4.2 mg/L; and serum vitamin C, 26.0 ± 6.4 μmol/L (see Table III).

Correlations between serum antioxidant vitamin levels and the other cardiovascular risk factors investigated in this study are shown in Table IV. However, no significant correlations were found between serum antioxidant vitamin levels and the other cardiovascular risk factors investigated (see Table IV).

Table I: Age and anthropometric measurements of participants

	Men with CHD (n:35)			Men without CHD (n:31)			
	$\bar{x}$ ± SD	Min	Max	$\bar{x}$ ± SD	Min	Max	p
Age (year)	54.2 ± 8.0	40.0	65.0	49.7 ± 5.8	40.0	64.0	0.013*
Height (cm)	170.6 ± 6.6	153.0	183.0	170.9 ± 6.5	160.0	184.0	0.836
Body Weight (kg)	81.8 ± 13.9	59.0	120.0	80.1 ± 8.1	68.1	95.1	0.877
BMI (kg/m^2)	27.9 ± 4.2	20.7	37.6	27.4 ± 2.6	23.3	31.9	0.614
BMH (kcal)	1718.7 ± 232.3	1330.0	2414.0	1724.3 ± 144.6	1499.0	2042.0	0.908
FM (kg)	24.8 ± 8.2	13.1	49.7	20.3 ± 4.4	10.3	31.8	0.021*
FM %	28.5 ± 4.9	17.2	42.4	25.2 ± 4.1	15.1	35.3	0.003*
FFM	56.3 ± 13.3	13.3	85.7	59.9 ± 5.7	49.3	71.0	0.167
FFM %	70.8 ± 6.8	41.0	82.8	74.2 ± 6.4	45.8	84.9	0.003*
BW (L)	42.7 ± 6.0	34.5	62.5	43.8 ± 5.0	35.3	58.9	0.235
BW %	53.3 ± 5.3	39.2	65.5	54.4 ± 3.7	42.3	62.1	0.332

BMI: Body mass index, BMH: Basal Metabolic Rate, FM: Fat Mass, FFM: Fat-Free Mass, BW: Body Water
* $p < 0.05$

Table II: Comparison of participants according to serum lipid profile

	Men with CHD (n:35)			Men without CHD (n:31)			
	$\bar{x}$ ± SD	Min	Max	$\bar{x}$ ± SD	Min	Max	p
TC (mg/dL)	165.9 ± 41.6	107.0	269.0	178.6 ± 22.6	118.0	205.0	0.135
TG (mg/dL)	156.1 ± 103.2	34.0	491.0	136.7 ± 44.8	56.0	211.0	0.802
HDL-C (mg/dL)	42.5 ± 12.3	7.0	65.0	49.8 ± 11.4	34.7	80.0	0.015*
LDL-C (mg/dL)	95.2 ± 39.7	44.8	197.8	96.6 ± 20.1	55.8	126.0	0.150
VLDL-C (mg/dL)	31.1 ± 19.3	12.0	98.2	32.3 ± 14.6	11.2	61.2	0.445
TC/HDL-C	4.6 ± 3.2	2.1	21.0	3.7 ± 0.8	2.3	5.7	0.516

TC: Total cholesterol, TG: Triglyceride, HDL-C: High-density lipoprotein-cholesterol, LDL-C: Low-density lipoprotein-cholesterol.
* $p < 0.05$

Table III: Comparison of participants according to dietary antioxidant vitamin intakes and serum levels

	Men with CHD (n:35)			Men without CHD (n:31)			
	$x \pm SD$	Min.	Max.	$x \pm SD$	Min.	Max.	p
Dietary Antioxidant Vitamin Intake							
Vitamin A (mcg)	1230.5 ± 711.5	334.4	4215.3	3137.8 ± 9779.2	473.4	54779.3	0.492
Vitamin C (mcg)	126.4 ± 64.4	8.1	306.1	114.8 ± 73.5	27.4	369.3	0.275
Vitamin E (mcg)	22.8 ± 15.5	3.6	77.8	14.6 ± 7.8	5.3	38.8	0.016*
Carotene (mcg)	3.4 ± 2.6	0.5	11.4	3.4 ± 2.6	0.3	13.0	0.969
Serum Antioxidant Vitamin Level							
Vitamin A (mg/L)	0.6 ± 0.2	0.30	1.1	0.8 ± 0.4	0.4	1.6	0.024*
Vitamin E (mg/L)	14.0 ± 4.2	7.31	31.2	17.2 ± 7.1	4.3	34.8	0.036*
Vitamin C (μmol/L)	26.0 ± 6.4	13.2	39.5	34.7 ± 9.2	20.8	53.9	0.000*

* $p < 0.05$

Table IV: The relation between the serum antioxidant vitamin levels and other risk factors for CHD in men with CHD

Risk Factors for CHD	Serum Vitamin A Level		Serum Vitamin E Level		Serum Vitamin C Level	
	r	p	r	p	r	p
Age	–0.200	0.256	–0.236	0.179	0.095	0.593
Body weight	–0.119	0.503	0.125	0.481	–0.073	0.698
FM	–0.184	0.298	0.089	0.616	–0.112	0.523
BMI	0.001	0.994	0.257	0.143	–0.243	0.166
TC	–0.155	0.381	0.085	0.631	–0.285	0.103
TG	–0.165	0.350	0.071	0.691	–0.244	0.165
LDL–C	–0.030	0.804	0.092	0.604	–0.118	0.505
HDL–C	–0.150	0.397	–0.164	0.353	–0.207	0.241
Dietary intake of vitamins	0.096	0.588	0.355	0.040	0.311	0.073

BMI: Body mass index, FM: Fat Mass, TC: Total cholesterol, TG: Triglyceride, HDL-C: High-density lipoprotein-cholesterol, LDL-C: Low-density lipoprotein-cholesterol.

Discussion

Development of CHD does not depend on any one factor, and in the etiology of CHD, several risk factors have been reported to play important roles [14]. In fact, to date, more than 200 risk factors associated with CHD have been identified [5]. Among these, obesity appears to play an important role and is thought to be one of the major risk factors associated with the development of CHD [15]. In support of this, every 3-unit increase in BMI is associated with a 45 % greater chance of developing CHD in men, and a 66 % greater chance of CHD in women [16]. Moreover, every 2-unit (kg/m^2) decrease in BMI reduces the risk of CHD development by 12 % [17]. Harris *et al.* [8] reported that individuals with BMI levels $\geq 27\ kg/m^2$ have a greater chance of developing CHD. Similarly, Iwao et al. [18] found that individuals whose BMI levels are $\geq 30\ kg/m^2$ are at increased risk for CHD. In contrast, in our study, mean BMI levels in men with CHD and men without CHD were not significantly different ($27.9 \pm 4.2\ kg/m^2$ and $27.4 \pm 2.6\ kg/m^2$, respectively), and no significant differences were found between men with CHD and men without CHD with respect to body weight ($p > 0.05$). In several studies, it has been reported that FM could be another risk factor for CHD [19–21]. For example, the correlation between FM and CHD was investigated in 21,925 males (30–83 years of age), and high FM and low FFM were identified as important risk factors for CHD development

[19]. In agreement with these results, in the present study we found that men with CHD had higher FM and FM% than men without CHD ($p<0.05$).

The role of cholesterol in CHD development was first reported in 1913. Since then, transport of cholesterol in the circulatory system was determined to be the most important factor in the development of CHD [4, 14]. Although serum TC levels should be less than 200 mg/dL (according to a Risk Factor Study), approximately 25 % of the Turkish community (approximately 7.5 million people) have higher TC levels [3]. Serum lipoprotein cholesterol levels are another risk factor that influences CHD development. In one study, CHD risk factors were investigated in 766 individuals and study participants were evaluated over a period of 8.7 years. At the end of the study, individuals with LDL-C cholesterol levels >122 mg/dL were found to have a 59 % greater chance of developing CHD. In addition, HDL-C levels <33 mg/dL were correlated with a 41 % increased risk for CHD [22]. In the present study, lipid profiles of study participants were examined, but only serum HDL cholesterol levels were found to be significantly different between the two groups. The mean HDL cholesterol levels in men with CHD and without CHD were 42.5 ± 12.3 mg/dL and 49.8 ± 11.4 mg/dL, respectively ($p<0.05$). In the National Cholesterol Education Program (NCEP) adult treatment panel, serum HDL-C levels lower than 40 mg/dL were identified as a risk factor for CHD [23]. Turkish society in general has been characterized as having low HDL-C levels, and at least 70 % of Turkish males have HDL cholesterol levels under 40 mg/dL [24].

There is also strong evidence that free radicals and other oxidants are important risk factors for atherosclerosis. Antioxidants reduce the levels of the superoxide anion and hydrogen peroxide, and keep hydroxyl radical levels low. Thus, they repress LDL oxidation and endothelial dysfunction, and the increased inflammatory responses associated with the atherosclerotic process. For these reasons, maintaining ideal serum levels of antioxidant vitamins by adequate antioxidant intake plays an important role in reducing the effects of oxidants [6, 7, 24–26]. When antioxidant vitamin intake was investigated in the present study, we observed that daily vitamin E intake in men without CHD was lower than in men with CHD ($p<0.05$), while other antioxidant vitamin intake levels were found to be similar between the groups. In contrast with our results, it is generally accepted that low vitamin E intake levels are associated with increased risk for CHD. However, the antioxidant activity of vitamin E does not only depend on dietary intake. Personal characteristics, genetic diversity, the degree of lipidemia, and biochemical events such as redox balance and oxidative stress can also affect the antioxidant activity of vitamin E [7].

We also examined serum antioxidant vitamin levels, and serum vitamin A, C, and E levels were found to be lower in men with CHD than in men without CHD ($p<0.05$). Whereas the average serum vitamin A, vitamin E, and vitamin C levels in men with CHD were 0.6 ± 0.2 mg/L, 14.0 ± 2.4 mg/L, and 26.0 ± 6.4 μmol/L, respectively, these values in men without CHD were found to be 0.8 ± 0.4 mg/L, 17.2 ± 7.1 mg/L, and 34.7 ± 9.2 μmol/L, respectively. These results are in agreement with previous studies [27–29]. For example, Riccione *et al.* [27], in a study of 232 individuals diagnosed with atherosclerosis *vs.* 291 healthy control individuals, found that serum vitamin E and A concentrations in the healthy subjects were higher. In addition, Gale *et al.* [28] found that low serum vitamin C, vitamin E, and beta-carotene levels were associated with increased risk for stenosis formation in 2888 males, aged 66–75. In another study, antioxidant profiles of 25 males and 13 females with ischemic stroke were compared with a control group (containing 37 members), and serum vitamin A and vitamin C levels in the control group were found to be higher than in the ischemic stroke group ($p<0.001$) [29].

In the present study, we also attempted to determine if there was any correlation between serum antioxidant vitamin levels and other CHD risk factors, but no significant correlation was found. Thus, on the basis of these results, serum antioxidant vitamin levels may be an independent risk factor for CHD.

Conclusion

Based on the results of this study, high body-fat mass and low serum antioxidant vitamin levels are important risk factors for CHD in adult males. In particular, body weight control and a balanced, adequate diet can minimize the risk for CHD in the general population.

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