Original Article

J Babol Univ Med Sci
Vol 17, Issu 4; Apr 2015; P:7-17

The Effect of Omega-3 on the Level of Serum Lipid Profile, Adipocytokines, and Indicator of Vascular Inflammation in Patients Diagnosed with Myocardial Infarction

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Received: Jun. 22th 2014 - Revised: Sep. 24th 2014 - Admitted: Feb. 4th 2015

ABSTRACT

BACKGROUND AND OBJECTIVE: Changes in inflammatory mediators have an important role in myocardial infraction. Since the anti-inflammatory effects of Omega-3 fatty acids on cardiovascular diseases was reported, this study was done to evaluate the Omega-3 effect on serum lipid profile, leptin, adiponectin, and E-selectin in patients with myocardial infraction.

METHODS: In this double blinded clinical trial study, 42 patients with myocardial infraction were randomly divided into two mediator and control group. For 10 weeks, the mediator group received 3 capsules of 1 gram Omega-3 daily, and the control group received 3 capsules of placebo (paraffin) daily. Concentration of serum lipid profile, leptin, adiponectin, and E-selectin was measured and compared at the beginning and at the end of the test. IRCT2012070410181N1

FINDINGS: At the end of the study, after comparing the Omega-3 receiving group with placebo group, there was a significant decrease of serum level of triglyceride (120.04±53.24 versus 150.76±48.84, p=0.021), leptin (6.92±2.71 versus 9.05±2.66, p=0.007), and E-selectin (20.98±10.04 versus 6.55±4.12), and there was a significant increase in the serum level of adiponectin (7.24±3.50 versus 6.55±4.12, p=0.026) in patients with myocardial infraction. In both groups, improvement in the size of blood lipids (TC, LDL-C, HDL-C) was obvious when compared to the primary sizes ($p<0.05$); but the difference between the two complementary and placebo group was not statistically significant.

CONCLUSION: The results show that receiving Omega-3 on a daily basis can modulate inflammatory factors in patients with myocardial infraction.

KEY WORDS: Omega-3, myocardial infraction, adiponectin, leptin, E-selectin, lipid profile.

Please cite this article as follows:

Haidari F, Tavakoli M, Heybar H, Helli B, Mohammadshahi M. The Effect of Omega-3 on the Level of Serum Lipid Profile, Adipocytokines, and Indicator of Vascular Inflammation in Patients Diagnosed with Myocardial Infarction. J Babol Univ Med Sci. 2015;17(4):7-17.

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Introduction

Myocardial Infraction (MI) is one of the common reasons of death and disability all around the world, which can lead to serious functional and mental changes in patients (1). Recent studies indicate wide range

of inflammatory changes in patients, in a way that high level of inflammatory factors such as C-Reactive Protein (CRP), cytokines and intercellular adhesion molecules were reported in patients with MI. Researchers believe that the increase of IL-6 (Interleukin=6), CRP, and E-selectin have an important role on the progress of MI (2 and 3). In contrast, empirical studies suggest that adiponectin has an anti-inflammatory and protective role against cardiovascular diseases (4). According to some of the researches, the levels of adiponectin decrease in patients with MI, and this reduction is in relation to the increase of inflammatory factors' density such as IL-6 or CRP (5 and 6). In return, leptin is a peptide hormone secreted by white adipose tissue, which has a role in causing MI in animal models by having mechanisms such as formation of blood clots, causing oxidative damage in heart endothelial cells, calcification of the vessel wall, and proliferation of vascular muscle cell (7). The increase of leptin also is reported to be an independent and dangerous factor in causing the first heart attack done by ischemia (6). On the other hand, based on the reports of epidemiological studies, the amount and type of consumed fats in regimen are closely related to lipid disorders, inflammatory changes, and vascular dysfunction in cardiovascular diseases; also it has been reported that receiving Omega-3 fatty acids decreases the level of triglycerides and blood pressure, increases fluidity of biological membranes, and prevents from vascular thrombosis and arrhythmias (8). Omega-3 fatty acids also have effects on lipoprotein metabolism, platelet function, endothelial function, inflammatory markers, production and secretion of pro-inflammatory cytokines, blood coagulation and fibrinolysis (9 and 10).

In addition, recently it has been suggested that consuming fish oil or its supplement can regulates the markers of vascular inflammation (11). However, according to the investigations, the results are contradictory especially on the effect of Omega-3 on the level of adiponectin, leptin, and E-selectin (12). There is little information about the effects of Omega-3 supplementation on markers of vascular inflammation such as adiponectin, leptin, and E-selectin in patients with myocardial infraction. Therefore, the present study is designed and conducted with the aim of evaluating the effects of Omega-3 supplementations on the serum lipid profile, adiponectin, and vascular inflammatory markers in patients with myocardial infraction.

Methods

Study design: this double-blinded clinical trial test with the registration number IRCT=2012070410181N1 was done on 42 patients with myocardial infraction in the heart section of Ahvaz's Imam Hospital. Sample size was calculated by considering the study of Mizia-Stec et al. (13) with 95% certainty and 90% power level and also by considering the possible loss of 21 people in each group. Sampling was done in an easy way. At the beginning of the research, all the subjects filled an informed consent after getting permission from the Ethics Committee of the Ahvaz University of Medical Sciences. The diagnosis criteria of MI were based on WHO criteria which include two symptoms of chest pain of angina characterized by low blood supply to the heart muscle that lasts at least 20 minutes, pathological changes indicative of ischemia / infarction on ECG waves (ST-segment elevation equal to or more than one mm in two contiguous leads), or more than two times increase of heart enzyme (1).

Patients who wished to participate in the study, with an age range of 45-65 years and being in a non-obese weight range (Body Mass Index = BMI<30) were enrolled, and in case of having liver problems, kidney failure, diabetes, and gastric problems, the use of antioxidant and Omega-3 supplements and consuming drugs that have effects on absorption and metabolism of Omega-3 were eliminated from the study at least 6 months before sampling. These patients were randomly divided into two groups of supplement and placebo. The patients in the supplement group received 3 capsules of 1-gram Omega-3 for 10 weeks. Each capsule consisted of 180 milligrams Eicosapentaenoic Acid (EPA) and 120 milligrams of Docosahexaenoic Acid (DHA). Patients of the placebo group received 3 capsules of placebo which contained 1 gram of paraffin and they seemed just like Omega-3 capsules. Supplement and placebo capsules were available to patients every two weeks, and during this time consuming placebo and Omega-3 supplements were weekly tracked with telephone calls. In the present study, placebo and Omega-3 supplements were provided by the medicinal company named Barij Essence.

Two under study groups were paired match for the kind of consuming drugs, gender, and BMI, and they were asked not to make any change in their regimen, physical activity, and drugs prescribed by their own doctor. Anthropometric data (weight, BMI, Waist to Hip Ratio=WHR), dietary intake, and physical activity were evaluated at the beginning and end of the study in both groups. In order to evaluate dietary intake of patients we used 3-hour food reminding questionnaire (including two work days and one holiday), and for food analyzing we used Nutritionist IV software. The level of physical activity was evaluated by International Physical Activity Questionnaire and calculated based on MET-minutes/week (14).

Biochemical evaluations: at the beginning of the study, 10 cc venous blood was taken after 12 hours of fasting from those hospitalized patients whose heart attack was 48 hours ago. Also at the end of the 10 weeks, blood sample was taken after fasting. To separate serum, blood samples were centrifuged for 5-10 minutes in the centrifugal with the speed of 3000 rpm, and were used for biochemical evaluation. Density of inflammatory factors was measured by using commercial kits and with ELISA (Enzyme-Linked Immunosorbent Assay) method.

In this way, the density of leptin, adiponectin, and E-selectin of serum samples were calculated considering the severity of their optical density at 450 nm by an ELISA reader, and by using standard curves and applying the dilution factors. In this study we used Orgenium kit made in Finland to calculate leptin and adiponectin, and Boster kit made in China to measure E-selectin. The levels of serum lipid profile (Triglyceride=TG, Total Cholesterol=TC, Low-density lipoprotein=LDL-C and High-density lipoprotein= HDL-C) were also measured enzymatically and by using Pars Azma kits made in Iran.

Statistical analysis: first, the normal distribution of all variables was evaluated by using the Kolmogorov-Smimov statistical test. The descriptive statistics were indicated for quantitative variables as mean and standard deviation. For comparing the mean of quantitative variables of two groups Independent Sample T-Test and in modified models (after the elimination of confounding variables) ANCOVA test was used.

Also for comparing the values before and after in each group, Paired Sample T-Test was used, and $p<0.05$ was considered significant.

Findings

There was no statistical difference between two supplement and placebo groups in regard to anthropometric features and level of physical activity before and after the study (table 1). Even after the modification of confounding variables (age, gender, calorie intake), these results were not significant.

However, intergroup comparison of the mentioned variables indicated that in both groups there was a significant reduction in the mean of weight, BMI, waist to hip, WHR, and the level of physical activity at the end of the study ($p<0.05$). In both groups, the intake of calorie, carbohydrate, fat, saturated fatty acid (SFA), Monounsaturated Fatty Acid (MUFA), and food cholesterol were significantly low at the end of the tenth month ($p<0.05$) (table 2).

Yet, after comparing two supplement and placebo groups, no significant statistical difference was seen in the amount of calorie intake at the beginning and end of the study.

The density mean of blood lipids was shown in both groups at the beginning and end of the study. At the beginning of the test, the levels of serum lipid profile TG, HDL-C, LDL-C, and TC had no significant difference between two groups of receiving Omega-3 supplements and placebo (table 3). The comparison of results of two groups at the end of the study suggested that Omega-3 supplement was able to decrease the serum level of TG significantly, when compared to placebo group ($p=0.039$). This effect was still standing after the modification of confounding variables ($p=0.021$).

At the end of the study it was seen that the serum levels TG, TC, and LDL-C were decreased in both groups, compared to the initial values, and density of HDL-C was increased ($p<0.001$). There was no significant difference in serum density of leptin, adiponectin, and E-selectin between two groups in the beginning of the study.

At the end of the study, after the modification of confounding variables, serum density of adiponectin was significantly higher in Omega-3 group ($p=0.026$) and leptin and E-selectin were significantly lower ($p=0.007$ and $p=0.011$, accordingly) (table 4).

Also in both groups there was a significant increase in the level of adiponectin serum and significant decrease in the level of leptin and E-selectin serum at the end of the study, when compared to the initial values ($p<0.05$).

Table 1. Comparison of the mean and standard deviation of anthropometric indexes and level of physical activity in patients with myocardial infarction receiving supplements of Omega-3 and placebo at the beginning and end of the study

Variable / Group	Supplement(N=21) Mean±SD	Placebo(N=21) Mean±SD	P_1	P_2
Weight (Kg)				
Beginning of study	81.42±18.83	80.91±14.73	0.731	0.624
End of study	79.90±18.44	78.42±13.90	0.771	0.411
P_3	**0.002**	**0.003**		
BMI (Kg/m^2)				
Beginning of study	29.30±3.74	28.79±3.75	0.663	0.508
End of study	28.46±3.62	28.38±3.52	0.729	0.669
P_3	**0.013**	**0.042**		
Waist (cm)				
Beginning of study	92.14±17.06	91.90±15.92	0.963	0.751
End of study	91.57±16.93	90.61±15.32	0.849	0.688
P_3	**0.039**	**0.000**		
Hip (cm)				
Beginning of study	108.33±11.47	108.61±10.58	0.934	0.843
End of study	107.71±11.27	107.54±10.52	0.961	0.894
P_3	**0.004**	**0.000**		
WHR				
Beginning of study	0.83±0.09	0.84±0.10	0.931	0.621
End of study	0.84±0.09	0.82±0.09	0.859	0.217
P_3	**0.044**	**0.011**		
Physical Activity (met/min/wk)				
Beginning of study	2451±359	2482±465	0.808	0.612
End of study	2155±295	2178±306	0.800	0.735
P_3	**0.001**	**0.002**		

P_1: comparison of the mean and standard deviation between the two groups of supplement and placebo; P_2: comparison of the mean and standard deviation between the two groups of supplement and placebo after modifying the effect of age, gender, calorie intake, and physical activity; P_3: comparison of the mean and standard deviation in both groups at the beginning and end of the study.

Table 2. Comparison of mean and standard deviation of dietary intake of the two Omega-3 supplement and placebo groups in patients with myocardial infarction at the beginning and end of study

Variable / Group	Supplement(N=21) Mean±SD	Placebo(N=21) Mean±SD	P_1	P_2
Calorie Intake (Kcal)				
Beginning of intervention	1987±814	2149±761		0.157
End of intervention	1790±546	1735±532	0.087	0.262
P_3	**0.021**	**0.000**	0.348	
Protein (gr)				
Beginning of intervention	69.15±29.09	76.75±27.18	0.387	0.272
End of intervention	73.86±22.54	75.48±23.17	0.819	0.625
P_3	0.226	0.678		
Carbohydrate (gr)				
Beginning of intervention	261.83±105.93	279.49±98.97	0.387	0.392
End of intervention	247.39±78.57	245.55±75.39	0.621	0.545
P_3	**0.014**	**0.006**		
Fat (gr)				
Beginning of intervention	70.81±29.78	80.00±28.33	0.312	0.121
End of intervention	55.50±16.94	51.29±15.74	0.408	0.198
P_3	**0.002**	**0.000**		
Saturated fatty acid (gr)				
Beginning of intervention	22.32±11.05	27.56±16.71	0.064	0.113
End of intervention	11.99±6.74	16.26±7.56	0.060	0.091
P_3	**0.013**	**0.001**		
Monounsaturated Fatty Acid (gr)				
Beginning of intervention	19.16±15.08	23.62±13.61	0.321	0.412
End of intervention	11.58±7.16	14.70±6.27	0.142	0.352
P_3	**0.005**	**0.003**		
Polyunsaturated fatty acids (gr)				
Beginning of intervention	17.25±8.37	19.47±8.07	0.114	0.138
End of intervention	11.83±7.20	15.24±7.63	0.130	0.146
P_3	0.059	0.051		
Cholesterol (mg)				
Beginning of intervention	278.69±74.41	283.73±70.75	0.310	0.271
End of intervention	141.05±80.66	136.03±69.00	0.829	0.397
P_3	**0.035**	**0.012**		

Table 3. Comparison of the mean and standard deviation of serum lipid profile in patients with myocardial infarction receiving supplements of Omega-3 and placebo at the beginning and end of the study

Group / Variable	Supplement(N=21) Mean±SD	Placebo(N=21) Mean±SD	P_1	P_2
TG (mg/d)				
Beginning of study	159.19±64.24	165.19±82.72	0.794	0.428
End of study	120.04±53.24	150.76±48.84	**0.039**	**0.021**
P_3	**0.000**	0.072		
TC (mg/dl)				
Beginning of study	210.33±54.06	198.90±43.92	0.457	0.337
End of study	167.42±46.78	174.90±33.81	0.556	0.213
P_3	**0.000**	**0.001**		
LDL-C (mg/dl)				
Beginning of study	140.06±44.35	128.72±32.19	0.349	0.214
End of study	101.99±43.22	103.62±31.37	0.889	0.712
P_3	**0.000**	**0.000**		
HDL-C (mg/dl)				
Beginning of study	38.42±5.14	37.14±3.42	0.346	0.190
End of study	41.42±4.69	39.52±4.37	0.182	0.068
P_3	**0.003**	**0.000**		

Table 4. Comparison of the mean and standard deviation of inflammatory markers in patients with myocardial infarction receiving supplements of Omega-3 and placebo at the beginning and end of the study

Group / Variable	Supplement(N=21) Mean±SD	Placebo(N=21) Mean±SD	P_1	P_2
Adiponectin (mg/L)				
Beginning of study	5.28±2.74	5.05±2.82	0.786	0.612
End of study	7.24±3.50	6.55±4.12	0.064	0.026
P_3	**0.001**	**0.028**		
Leptin (ng/ml)				
Beginning of study	14.22±4.79	15.24±3.85	0.078	0.062
End of study	6.92±2.71	9.05±2.66	**0.014**	**0.007**
P_3	**0.000**	**0.000**		
E-selectin (ng/ml)				
Beginning of study	39.67±21.79	37.24±16.04	0.683	0.592
End of study	20.98±10.04	27.06±12.58	0.058	**0.011**
P_3	**0.000**	**0.000**		

Discussion

The results showed that daily supplementation of 3-gram Omega-3 for 10 weeks can decrease serum level of triglyceride, leptin, and E-selectin and increase the serum level of adiponectin in patients with myocardial infraction. However, according to the results it was obvious that consuming Omega-3 supplements did not make any significant changes in anthropometric indices (weight, BMI, waist, hip, WHR), calorie intake, and macronutrients in those who received supplement in comparison with placebo group. These findings are in accordance with the results of Mori et al.'s study which evaluated the effect of receiving Omega-3 fatty acids for 6 weeks in people with high levels of serum lipid (15). In the study of Cussons et al. also the Omega-3 supplement did not make any significant change in anthropometric measures (16). Similar results were reported by Tsitouras et al. in non-obese elderly (17). However, the BMI of women with type-2 diabetes and

metabolic syndrome was decreased by consuming Omega-3 fatty acid supplements in the study of Hajianfar et al. and Ebrahimi et al. (18 and 19). It has been suggested that the differences between these results might be related to gender, age, and different BMIs of various people in the studies (18-21). Also it is possible that Omega-3 fatty acids cause reduction in BMI and body weight by various mechanisms like reducing calorie intake (22). Anyway, considering the fact that there have been no significant changes in the amount of calorie intake in neither of the groups at the end of the study, lack of significant changes in anthropometric indices of studied groups can be because of not having significant changes in calorie intake.

In the present study, the serum level of TG in the group receiving the Omega-3 supplement in comparison to the placebo group was significantly reduced at the end of the study. However, the Omega-3 supplement in comparison to placebo was not able to make any significant changes in other lipids. There is contradictory information at hand about the effects of Omega-3 fatty acids on lipid profile. Aligned with the results of this study, the supplement of Omega-3 fatty acids caused reduction in TG serum in the study of Cussons et al., but it did not make any significant changes in serum levels of total cholesterol, LDL-C, and HDL-C in women with polycystic ovary syndrome (16). Reduction of TG serum level because of Omega-3 fatty acid supplements was seen in other studies such as those on people with severe hyperlipidemia and diabetics (22 and 23). In the study of Nilsen et al. the Omega-3 supplement caused reduction in TG level and increase in HDL-C level in patients with myocardial infraction (24). It has been suggested that the effect of Omega-3 fatty acid on lipid profile, especially on TG serum level, depends on dose of use (19). Omega-3 fatty acids can make improvement in lipid patterns through different mechanisms. It has been shown that these fatty acids act as Peroxisome Proliferator Activated Receptors (PPAR). Activation of these receptors (specifically Gama receptors) increases the expression of genes encoding proteins involved in fatty acid oxidation in the liver and muscles; in contrast it inhibits the expression of genes involved in the synthesis (25). In addition, a part of Omega-3 fatty acid influences is applied through activating Adenosine Monophosphate-Activated Protein Kinase (AMPK). This enzyme acts as a metabolic sensor, and causes balance between cellular metabolic fuels such as balance between oxidation and fatty acid biosynthesis (26). Also long-chain Omega-3 fatty acids can improve the activities of low-density lipoprotein receptors (LDL-C) in liver (17). It is indicated in this study that intergroup comparison (comparing the measures before and after intervention) showed significant improvement in all the lipids at the end of the study. Therefore one of the possible reasons for the lack of significant effect of Omega-3 supplement (in comparison to placebo) on other lipids except for TG in this study can be because of concomitant use of lipid-lowering drugs (like Statins), and lifestyle modification (like losing weight) following the occurrence of MI in patients under study, since these factors have high effects on the improvement of lipids in patients and can cause the positive effects of Omega-3 on blood lipids to not be significant.

Our study showed that Omega-3 supplement increased the density of adiponectin in comparison to placebo, after it was taken for 10 weeks. The reduction of adiponectin level and increase of leptin density in obese people and those diagnosed with kinds of cardiovascular diseases have been reported in various studies (20 and 27). However, there is little information about the effects of Omega-3 on changes in adiponectin density (leptin and adiponectin) in patients with MI. Adiponectin is a protein derived from adipose tissue, which about 5-30 µg/ml of it exists in human serum (28). Our study showed that Omega-3 supplement in 10 weeks can significantly increase adiponectin density in comparison with placebo. However, there is little information about the effects of Omega-3 on changes of adipocytocins (leptin and adiponectin) in patients with MI. The results of our study were in accordance with several studies. Sneddon et al. indicated that the combination of conjugated linoleic acid and Omega-3 fatty acid supplementation prevents the increase of abdominal fat mass in obese people and increases lean body mass and plasma adiponectin levels (20). Kondo et al. indicated that taking Omega-3 for 8 weeks can lead to an increase in plasma adiponectin in non-obese women (29). In the studies conducted by Itoh et al. on obese people and Krebs et al. on diabetic women, the Omega-3 supplement increased plasma adiponectin (30 and 31). In general, high levels of adiponectin serum are considered as a protective adipocytocin, in relation to decreasing the dangers of myocardial infraction (27 and 28). Various studies showed that adiponectin leaves its tracks on liver and muscles through its AMPK activating mechanism. It has been reported that adiponectin can activate AMPK in endothelial cells,

followed by stimulation of nitric oxide (NO) in these cells and it eventually decreases the infraction in animal models. Therefore, activation of AMPK can be one of the mechanisms of beneficial and protective effects of adiponectin against cardiovascular diseases, especially myocardial infraction (32). On the other hand, it seems that Omega-3 has also a role in increasing the secretion of adiponectin by stimulating PPAR-gamma. So it can be concluded that Omega-3 can improve and decrease the inflammatory functions in these patients through increasing the levels of adiponectin serum (33). However, the lack of Omega-3 influence on the increase of adiponectin has been reported in some of the studies. Kratz et al. reported that consuming a diet rich in Omega-3 fatty acid (3.5% calorie intake) for 4 weeks did not make a significant increase in plasma adiponectin in healthy obese men and women (34). Also in the study of Mizia-Stec et al. consumption of 1-gram Omega-3 for 4 weeks did not have a significant effect on adiponectin serum density in patients with MI (13). It seems that lower dose and shorter duration of two recent studies compared to the present study, can have a role in explaining the contradictory findings. In general, high levels of adiponectin serum are considered as a protective adipocytocin, in relation to the danger of infraction (35,36).

Recently it has been suggested that leptin might have a role in regulating myocardial blood flow. The results of a few studies in this area show that the amount of leptin increases in myocardial infraction (37 and 38). Our study indicates that consuming Omega-3 supplement for 10 weeks leads to a significant increase in leptin density when compared to placebo. There is little information about the effects of Omega-3 on changes in leptin density in patients with MI. In accordance with our study, Olza et al. showed that diets containing eicosapentaenoic acid and docosahexaenoic acid for 6 months can improve TG of blood and decrease leptin serum in elderly (39). Mori et al. also indicated that consuming fish on a daily basis with taking Omega-3 supplements can effectively lead to weight loss than only lowering the level of leptin serum. These researchers suggested that simultaneous intake of Omega-3 fatty tissue and physical activity can increase adipose tissue lipolysis and decrease serum leptin levels (9). The effect of unsaturated fatty acids especially Omega-3 as a decreasing factor of plasma leptin level was also confirmed in other studies (40). Mostowik et al. also reported that consuming 1-gram Omega-3 for 30 days can significantly increase plasma adiponectin to leptin in patients waiting for the surgery (41). Also, Richelle et al. showed that feeding Omega-3 to rats for 8 weeks can decrease in leptin gene expression. According to the findings of these researchers, leptin gene expression is dependent on the type of consumed fat; a diet high in cholesterol decreases the expression of leptin, while a diet with high Omega-3 fatty acids can increase leptin gene expression. The mechanisms of the effects of dietary fat on leptin gene expression are unclear, but it seems that Omega-3 fatty acids have a role in increasing the expression of adiponectin through stimulating PPAR-gamma (42). Also it has been suggested that these fatty acids cause reduction in secretion of leptin by activating PPAR-gamma ligands (such as thiazolidinedione) (41). In contrast to these studies, Sneddon et al. did not find any changes in the amount of leptin in thin and obese men after receiving Omega-3 (20). Since no significant differences was seen in decrease of serum leptin levels in Omega-3 supplement at the end of the study when compared with the control group, this difference is due to the effect of Omega-3 supplement on the reduction of this factor's level.

In this study, the consumption of Omega-3 supplement lead to a significant reduction in E-selectin density after 10 weeks, when compared to placebo in people with myocardial infraction. The effect of Omega-3 fatty acids on cell adhesion molecules in patients with cardiovascular diseases was evaluated in a few studies. The results of previous studies show that increased plasma levels of E-selectin in patients with MI, indicates the increase of vascular endothelial cells' activity in response to inflammatory cytokines (43). Yusof et al. reported that daily consumption of 2 grams Omega-3 leads to a considerable decrease in E-selectin serum in patients waiting for heart surgery. These researchers suggested that Omega-3 fatty acids reduce the expression of adhesion molecules on endothelial cells by their inhibiting effect on production of pro-inflammatory cytokines such as TNF-α and IL-1 (44). In contrast, Egert et al. did not find any changes in their study on 40 healthy people with the mean age of 26 after 5 weeks after consuming DHA and EPA in E-selectin (40). It seems that choosing healthy people without cardiovascular risk factors, short period of research, and low mean of age in sampling in the recent study can explain the differences between the present study's findings and the this study. Generally and according to these results, consumption of 3 grams of Omega-3 a day for 10 weeks improves the level of blood TG in patients with MI. It can also compensate for decreased serum

levels of adiponectin and increased leptin and E-selectin levels in these patients. These effects support themodifying roles of Omega-3 fatty acids on vascular inflammation in MI. However, the need for further researches in this area is recommended.

Acknowledgement

We hereby appreciate the financial support of Nutrition and Metabolic Disorders Research Center, Ahvaz Jundishapur University of Medical Sciences and Research Council of Arvand International Division.

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