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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Supplementation with omega-3 acids after myocardial infarction and modification of inflammatory markers in light of the patients' diet: a preliminary study

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Abstract

Background: Neuroendocrine activation, activation of proinflammatory cytokines and platelets, and endothelial dysfunction play a significant role in the development of heart failure (HF).

Aim: The aim of the work was to assess the effect of supplementation with EPA and DHA in a daily dose of 1 g on selected inflammatory markers and platelet activation in patients with HF after recent myocardial infarction in light of their diet.

Methods: This preliminary study was a randomised, double-blind trial involving 30 patients with post-infarction HF. One group received a product containing 1 g of omega-3 acids, while the other received placebo, i.e. corn oil 1 g daily for 12 weeks. At baseline and at week 12, venous blood was obtained in the fasted state in order to determine the following parameters: NT-proBNP, fibrinogen, INR, creatinine clearance, serum lipid profile, hsCRP, troponin, glucose, transaminases, GGTP, MCP-1, pentraxin 3, and CD-40. To evaluate the patient's diet and dietary intake of omega-3 acids, a 24-h dietary interview and the Block's Food Frequency Questionnaire (FFQ) were applied.

Results: Supplementation of omega-3 acids in a dose of 1 g per day had no effect on lipid or inflammatory parameters, with the exception of pentraxin 3. In both groups, after three months of supplementation, overall consumption of energy and saturated fatty acids was significantly higher ($p < 0.05$).

Conclusions: Potential benefits associated with supplementation were nullified by a highly atherogenic diet. Apparently, supplementation of omega-3 acids without simultaneous dietary education and nutrition control does not bring the expected effect. Further research involving a larger group of patients is needed to better understand the relationship between patient's diet and the effectiveness of omega-3 supplementation.

Key words: cluster of differentiation 40 (CD-40), docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA), inflammatory markers, myocardial infarction, nutrition

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INTRODUCTION

Heart failure (HF) is a complex disease, involving a range of pathomechanisms. Neuroendocrine activation, activation of proinflammatory cytokines and platelets, and endothelial dysfunction play a significant role in development of HF [1]. With disease progression, inflammatory processes, damaging the blood vessel wall as a result of cytotoxic activity of cytokines, start to prevail and lead to increasing myocardial insufficiency. Those mechanisms have been confirmed in numerous studies in which increased expression of proinflammatory cytokines, such as tumour necrosis factor alpha (TNF-α), interleukin (IL)-6, and IL-18, as well as certain chemokines, e.g. monocyte

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chemotactic protein-1 (MCP-1), IL-8, CXCL16, CCL21, and pentraxin 3 (PTX3), was demonstrated in patients with HF [2]. Recently, the role of the CD-40-sCD-40L signalling pathway, being a common element of the mechanisms of activation of coagulation and inflammation, has raised much interest [3].

Pentraxin 3 is considered a good marker of inflammatory activity and stability of atherosclerotic plaques. Increased PTX3 expression in atherosclerotic arteries in comparison with non-atherosclerotic arteries was demonstrated using immunohistochemical methods [4]. According to certain authors, PTX3 may have a prognostic role in HF. Matsubara et al. [5] demonstrated that PTX3 is an independent marker of left ventricular (LV) diastolic dysfunction in HF with preserved ejection fraction (EF), and that myocardium may contribute to increased concentration of this protein in the blood.

Monocyte chemotactic protein-1 expression contributes to stimulation of inflammatory response, LV remodelling, and development of HF after myocardial infarction (MI). De Lemos et al. [6] demonstrated that MCP-1 is an independent diagnostic marker of the risk of acute incidents and chronic phase after acute coronary incidents. Studies by Hohensinner et al. [7] indicated that high MCP-1 concentrations were associated with increased all-cause mortality in patients with HF. The authors also suggested that cytokines, including MCP-1, may play a major role in progression of cardiomyopathy [7]. Certain studies in animals indicated that inhibition of MCP-1 production might prevent development of post-infarction HF [8]. Studies by Stumpf et al. [9] demonstrated that increased concentration of MCP-1 in patients with HF was largely due to platelet activation via a mechanism involving CD-40-CD154 receptors.

Study results concerning increased cluster of differentiation 40 (CD-40) concentration in patients with HF are inconsistent. In certain, but not all, studies, increased levels of soluble CD-40L were found in HF, while in other studies, increased levels of CD-40L were noted only if measured on the platelet surface or in platelet lysates [10]. Non-pharmacological interventions, including those concerning dietary changes, may potentially modify functioning of blood vessels and vascular endothelium, and activate pro-inflammatory cytokines. The results of numerous prospective studies indicate that the nutrients characteristic for the Mediterranean diet that most effectively reduce cardiovascular mortality and may affect mechanisms associated with inflammation may be certain omega-3 acids (i.e. eicosapentaenoic acid [EPA] and decosahexaenoic acid [DHA] [11].

The DART study demonstrated that the use of a diet rich in sea fish-derived omega-3 acids for two years in patients after MI resulted in overall mortality reduction by 29% in comparison with the control group [12]. In the GISSI study concerning secondary prevention in patients after recent MI, an intervention lasting 3.5 years resulted in a significant reduction of cardiovascular endpoints by 20% and cardiovascular mortality by 30% [13]. The results of those studies were not confirmed by later works, including the OMEGA study, in which no benefits attributable to omega-3 acids supplementation were noted [14]. Therefore, it is necessary to conduct further studies in order to clarify these discrepancies, determine the factors affecting the efficacy of supplementation, and fully explain the mechanisms of potentially beneficial activity of omega-3 acids.

The aim of the work presented here was to assess the effect of supplementation with EPA and DHA in a daily dose of 1 g (as recommended by the Polish Society for Atherosclerosis Research and the Polish Society of Cardiology) on selected inflammatory markers and platelet activation in patients with HF after recent MI in light of their diet.

METHODS

The study was a randomised, double-blind trial involving 30 patients with post-infarction HF. The patients were recruited according to the following inclusion criteria: age ≥ 18 years (men and women), post-infarction HF with LVEF 40 ± 5%, New York Heart Association (NYHA) class II–III, optimum treatment according to current standards (acetylsalicylic acid, an angiotensin converting enzyme inhibitor, a beta-adrenolytic agent, a statin, an aldosterone receptor blocker), 6–12 weeks after MI. The following exclusion criteria were applied: haemodynamic instability, non-ischaemic HF (of other aetiology), hypersensitivity to the applied products, neoplastic diseases or other serious conditions precluding long-term follow-up, uncontrolled diabetes, severe liver damage, end-stage kidney disease, the use of products containing polyunsaturated fatty acid in the previous three months, or high dietary intake of omega-3 acids. On discharge from the hospital, all enrolled patients were provided only with general information concerning the Mediterranean diet and the necessity to use it, without specific education by a dietician. Clinical characteristics of patients in both groups are presented in Table 1.

The patients were randomly assigned to one of two groups. One group received a product containing 1 g of omega-3 acids (two capsules), while the other received placebo, i.e. corn oil 1 g daily (two capsules) for 12 weeks.

One capsule of the omega-3 product contained 846 mg of fish oil, including 510 mg of omega-3 acids (179 mg of DHA and 255 mg of EPA).

Fourteen patients in each group completed the study; one person died due to an acute cardiovascular event, and another withdrew their consent during the study.

At baseline and at week 12, venous blood was obtained in the fasted state in order to determine the following parameters: N-terminal pro-B-type natriuretic peptide (NT-proBNP), fibrinogen, international normalised ratio (INR), creatinine clearance, serum lipid profile, high sensitivity C-reactive protein (hsCRP), troponin, glucose, transaminases, gamma-glutamyl transpeptidase (GGTP), MCP-1, PTX3, and

Table 1. Characteristics of the study groups

	Omega-3 group (n = 15)	Placebo group (n = 15)	P
Age [years]	67.5 ± 0.70	62.06 ± 1.41	NS
Sex: men	13 (86%)	13 (86%)	NS
Body mass index [kg/m^2]	29.97 ± 3.68	26.22 ± 2.53	0.008
Smokers	4 (27%)	5 (33%)	NS
NYHA class II/III	11 (73%)/4 (27%)	12 (80%)/3 (20%)	NS
Left ventricle [%]	42.4 ± 0.02	40.6 ± 0.03	NS
Diabetes mellitus	3 (20%)	3 (20%)	NS
Hypertension	7 (47%)	12 (80%)	NS
The start time of the study (days after infarction)	61.9 ± 21.8	58.6 ± 19.7	NS
Treatment:			
Beta-blocker	15 (100%)	14 (93%)	NS
ACEI	13 (86%)	15 (100%)	NS
Statin	15 (100%)	15 (100%)	NS
ASAs	15 (100%)	15 (100%)	NS
Clopidogrel	15 (100%)	15 (100%)	NS
ARB	2 (13%)	6 (40%)	NS
IPP	13 (86%)	14 (93%)	NS
Insulin	1 (7%)	2 (14%)	NS

Data are presented as mean ± standard deviation or number (percentage); ACEI — angiotensin-converting-enzyme inhibitor; ARB — angiotensin receptor blockers; ASA — acetylsalicylic acid; IPP — proton pump inhibitors; NYHA — New York Heart Association

CD-40. Laboratory tests were performed using enzymatic methods, with ready-to-use kits or ELISA tests.

In order to evaluate the patient's diet and dietary intake of omega-3 acids, a 24-h dietary interview and the Block Food Frequency Questionnaire (FFQ) with respect to omega-3 acids were applied. Each patient completed the diet interview at baseline (i.e. on the day on which supplementation was started) and after 12 weeks. The nutritional value of the diet was calculated using "Dieta 5" software developed by the Food and Nutrition Institute.

The study was approved by the Bioethics Committee of the Medical University of Warsaw.

Statistical analysis

All continuous variables were presented as arithmetic mean value ± standard deviation and compared using a test based on the t-Student distribution. The effect of administration of the omega-3 product or placebo on selected biochemical parameters was analysed using a univariate ANOVA test. The frequency of consumption of specific food products was compared between the groups using the non-parametric Mann-Whitney U test for variance. Statistical analysis was performed using Statistica 10.0 PL software.

RESULTS

Baseline evaluation of diet demonstrated previous abnormal proportions of the consumed fatty acids; those abnormalities, in particular the PUFA/SAFA ratio, worsened in the course of the study. In the omega-3 group, the PUFA/SAFA ratio decreased from 0.46 to 0.26 during the study; in the placebo group, the reduction was from 0.76 to 0.54. In both groups, the daily dietary intake of omega-3 acids decreased: from 0.87 ± 0.02 g to 0.74 ± 0.37 g in the investigated group and from 0.76 ± 0.26 g to 0.668 ± 0.44 g in the placebo group; the differences were not statistically significant. Statistical analysis demonstrated significant differences in increased overall consumption of energy and saturated acids in both groups in relation to baseline values ($p < 0.05$). The results of 24-h dietary interview were confirmed by those of the food frequency assessment. Consumption of selected nutrients at baseline and after completion of the study is presented in Table 2, and the results concerning the frequency of consumption of specific food product groups are shown in Table 3.

Table 4 presents the mean values of biochemical parameters in both groups. ANOVA analysis demonstrated the effect of the applied product only on the change of PTX3 concentration. No such effect was observed for the other inflammatory markers, i.e. CRP, MCP-1, homocysteine, or CD-40. The results of ANOVA analysis are presented in Table 5.

DISCUSSION

In the guidelines of the European Society of Cardiology (ESC) concerning treatment of HF, supplementation of polyunsaturated omega-3 fatty acids is listed among the recommended therapies. This is a class IIb recommendation (level of evidence: B). Among the recommendations of the ESC

Table 2. Consumption of energy and nutrients in both patient groups before the start and after completion of dietary supplementation

	Omega-3 group		Placebo group	
	Baseline	After	Baseline	After
Energy [kcal]	1793.50 ± 1286.2	2373.00 ± 1169.55[a]	1529.8 ± 469.31	1824.2 ± 457.78[a]
% kcal of protein	19.90 ± 3.11	21.90 ± 4.10	19.16 ± 8.14	21.11 ± 7.92
% kcal of fat	31.75 ± 12.94	31.50 ± 10.60	31.30 ± 6.402	30.70 ± 7.24
% kcal of carbohydrates	48.40 ± 9.75	48.60 ± 7.49	49.53 ± 12.69	50.13 ± 10.68
SAFA [g]	27.34 ± 27.49	34.70 ± 31.5[a]	19.09 ± 9.83	24.98 ± 11.33[a]
SAFA [% diet energy]	8.88 ± 6.05	11.62 ± 6.23	10.41 ± 3.57	11.22 ± 3.61
MUFA [g]	30.76 ± 34.27	33.99 ± 39.89	23.28 ± 9.97	23.37 ± 9.59
MUFA [% energy]	12.47 ± 8.25	10.43 ± 9.99	13.69 ± 4.35	13.06 ± 4.38
PUFA [g]	10.84 ± 11.49	8.50 ± 8.06	14.31 ± 11.99	13.01 ± 11.28
PUFA [% energy]	4.54 ± 2.51	2.81 ± 1.67	8.88 ± 8.44	7.61 ± 7.56
PUFA [g]	11.86 ± 11.49	9.52 ± 8.06	14.48 ± 11.39	13.57 ± 11.28
PUFA [% energy diet + supplement]	5.09 ± 2.51	3.27 ± 1.67	8.98 ± 8.44	7.93 ± 7.56
Omega-3 from diet [g]	0.87 ± 0.02	0.74 ± 0.37	0.757 ± 0.26	0.668 ± 0.44
Omega-3 from diet + supplement [g]	1.89 ± 0.02	1.76 ± 0.37	0.767 ± 0.26	0.678 ± 0.44
Cholesterol [mg]	337.50 ± 391.03	371.50 ± 245.36	216.93 ± 175.78	307.6 ± 214.82
Dietary fibre [g]	16.20 ± 4.38	15.85 ± 1.20	15.46 ± 8.62	14.7 ± 7.40
PUFA/SAFA ratio diet	0.39	0.24	0.74	0.52
PUFA/SAFA ratio diet + supplement	0.43	0.26	0.76	0.54

Data are presented as mean ± standard deviation; [a]$p < 0.05$ after supplementation vs. baseline; MUFA — monounsaturated fatty acid; PUFA — polyunsaturated fatty acid; SAFA — saturated fatty acid; PUFA/SAFA ratio — polyunsaturated fatty acid/saturated fatty acid ratio

Table 3. Selected products and the frequency of their consumption (% of the group) based on the Food Frequency Questionnaire (FFQ)

	Omega-3 group		p	Placebo group		p
	Baseline	After		Baseline	After	
Wholemeal bread everyday	11 (73.3%)	6 (40%)	0.220	10 (66%)	6 (43%)	0.282
Groats, flakes, muesli ≥ 2–3 × week	5 (33%)	2 (14.2%)	0.256	5 (33%)	3 (21.4%)	0.128
Legumes > 1 time a week	5 (33%)	5 (35.7%)	0.946	1 (6.6%)	1 (7.1%)	0.961
Fishes at least once a week	10 (66%)	5 (35.7%)	0.267	10 (66%)	7 (50%)	0.181
Butter spreads everyday	5 (33%)	10 (71%)	0.112	4 (26.6%)	9 (64.2%)	0.146
Margarine spreads everyday	10 (66%)	5 (35.7%)	0.112	9 (60%)	4 (28.5%)	0.145
Meat everyday	6 (40%)	12 (85%)	0.05	5 (33%)	8 (57.1%)	0.235
Vegetables everyday	7 (10%)	3 (21.4%)	0.223	4 (26.6%)	3 (21.4%)	0.771
Fruit everyday	3 (20%)	1 (7.1%)	0.221	1 (6.6%)	1 (7.1%)	0.976

Data are presented as number (percentage)

concerning treatment of HF, this one is the least commonly applied in Poland, although the consumption of omega-3 acids and sea fish in Poland is very low. The WOBASZ studies demonstrated that in Poland, the level of consumption of fish, the main source of omega-3 acids, is among the lowest in the European Union — 15–16 g per day per adult individual, i.e. half the recommended value [15]. In this preliminary study, we wanted to find out what effect supplementation of omega-3 acids at the most commonly recommended level of 1 g per day would have on selected biochemical parameters, especially inflammatory markers, with standard application of non-pharmacological interventions concerning diet.

Table 4. Mean values of biochemical parameters at baseline and after completion of supplementation in both study groups

	Omega-3 group		Placebo group	
	Baseline	After	Baseline	After
AST	24.5 ± 7.39	26.84 ± 8.2	29.5 ± 12.9	24.53 ± 8.7
ALT	34.57 ± 22.6	34.76 ± 23.4	34.6875 ± 20.6	32.2 ± 15.2
GGTP	65.3 ± 88.6	64.76 ± 116.0	41.5 ± 37.7	47.53 ± 59.2
Glucose	118.5 ± 32.3	115.69 ± 21.7	115.18 ± 28.1	117.66 ± 29.2
Creatinine	0.86 ± 0.13	0.89 ± 0.13	0.905 ± 0.17	0.898 ± 0.19
Troponin I	0.042 ± 0.08	0.030 ± 0.05	0.031 ± 0.07	0.022 ± 0.05
BNP	950.21 ± 1124.5[a]	493.15 ± 560.8	910.56 ± 671.5	436.86 ± 365.5[a]
CRP	2.73 ± 2.3	2.72 ± 4.4	3.04 ± 3.0	3.28 ± 4.4
TC	136.64 ± 27.9	139.84 ± 22.4	132.31 ± 33.3	135.2 ± 33.9
Triglyceride	93.14 ± 30.1	105.76 ± 51.2	102.43 ± 39.3	104.73 ± 43.3
HDL-C	53.35 ± 13.8	53.38 ± 13.2	45.81 ± 12.2	48.06 ± 13.1
LDL-C	64.71 ± 23.8	65.30 ± 17.3	65.87 ± 24.7	66.33 ± 27.2
Pentraxin	3.154 ± 1.69	3.021 ± 1.3	2.86 ± 1.75	5.09 ± 3.1[a]
MCP-1	191.15 ± 45.6	187.15 ± 1.08	223 ± 105.7	222.26 ± 2.21
CD-40	551 ± 130.7	558.5 ± 145.7	651.4 ± 259.4	688.64 ± 79.6
Homocysteine	13.44 ± 7.08	13.51 ± 8.1	10.33 ± 4.3	10.09 ± 3.8

Data are presented as mean ± standard deviation; [a]$p < 0.05$; ALT — alanine transaminase; AST — aspartate transaminase; BNP — B-type natriuretic peptide; CD-40 — cluster of differentiation 40; CRP — C reactive protein; GGTP — gamma-glutamyl transpeptidase; HDL-C — high-density lipoprotein cholesterol; LDL-C — low-density lipoprotein cholesterol, MCP-1 — monocyte chemotactic protein-1; TC — total cholesterol

Table 5. ANOVA analysis for the effect of supplementation on selected biochemical parameters

	Omega-3 group Mean changes ± SD	Placebo group Mean changes ± SD	F	P
C reactive protein	0.123 ± 3.94	0.186 ± 4.54	0.001158	0.973
Pentraxin	–0.133 ± 1.201	2.232 ± 2.56	9.029926	0.005
Monocyte chemotactic protein-1	–4 ± 39.43	–0.733 ± 59.88	0.027807	0.868
Cluster of differentiation 40	7.230 ± 113.34	37.2 ± 248.11	0.733214	0.399
Homocysteine	0.069 ± 5.22	–0.242 ± 4.67	0.028093	0.868

Data are presented as mean ± standard deviation (SD)

The study demonstrated no significant effect of supplementation on the level of lipids or homocysteine; an increase in concentration of triglycerides in the investigated group was surprising although not statistically significant. The study demonstrated no effect of supplementation on CRP concentration. This might have been due to the fact that patients with relatively low baseline hsCRP concentrations (the mean concentration was 2.73 and 3.04 in the omega-3 group and the placebo group, respectively) were enrolled, haemodynamically stable and receiving optimum treatment according to the ESC standards; therefore, additional supplementation of fish oil had little effect on CRP concentration. Consistent evidence may be found in the literature. The effect of supplementation of fish oil on the level of hsCRP in patients with HF was investigated in four independent studies, and none of them demonstrated significant changes in this parameter [10].

De Lamos et al. [16] demonstrated that MCP-1 concentration values over 238 pg/mL might be associated with increased mortality in patients with ischaemic heart disease. It is known that the Mediterranean diet, rich in omega-3 fatty acids, may contribute to a reduction in the MCP-1 level. Based on in vitro studies, Diaz Encarnacion et al. [17] stated that EPA and DHA inhibited MCP-1 production; this may be an important mechanism of well documented anti-inflammatory properties of this group of fatty acids.

Unfortunately, not many clinical trials evaluating the effect of supplementation of omega-3 acids on the concentration of MCP-1 have been conducted so far. In the study discussed

here, no significant effect of supplementation on the concentration of MCP-1 was found. The study by Darghosian et al. [18] also demonstrated no such effect, although EPA and DHA were used in a dose of 4 g per day.

The lack of effect of supplementation was also noted with respect to the CD-40 ligand. Din et al. [19] demonstrated that supplementation of omega-3 fatty acids had no effect on vascular endothelial function or platelet activation in patients after MI. Similarly, no changes in the expression of CD-40 or the concentration of the CD-40 ligand were found. The lack of effect of supplementation of fish oil or omega-3 acids was also confirmed in other studies, although Aarsetoy et al. [20] used a high dose of EPA and DHA (4 g in total) for a long time (12 months).

Pentraxin 3 is increasingly considered a sensitive inflammatory marker. According to studies, its plasma level is very low in healthy individuals but increases rapidly (to 200–800 ng/mL in 6–8 h) as a result of activity of inflammatory mediators, e.g. in endotoxic shock, sepsis, and MI [21].

Suzuki et al. [22] observed increased PTX3 concentration in patients with HF in comparison with healthy individuals (the upper limit of concentration in healthy subjects was 4 ng/mL), and the level of PTX3 increased with the progression of HF (to a higher NYHA class); therefore, the suspicion that it is a good marker of HF progression may be justified. In addition, the same study demonstrated that acute cardiac events were more common in patients with high PTX3 plasma concentration than in patients with normal PTX3 level [22]. PTX3 is also considered a good early marker of acute MI [23]. In our study, low baseline levels of PTX3 were observed both in the omega-3 group and the placebo group. ANOVA analysis demonstrated that the only parameter on which the product used for supplementation had any effect was PTX3 concentration. It may be supposed that supplementation of omega-3 fatty acids prevented the increase in PTX3 concentration over 5 ng/mL observed in the placebo group receiving corn oil. It should also be noted that with time, both in the omega-3 group and the placebo group, the principles of the Mediterranean diet were less and less strictly observed by the patients; this might have some effect on PTX3 concentration. The effect of omega-3 acids on the PTX3 level was observed in the GISSI-Heart Failure Trial, in which an increase in the EPA level over several months due to daily supplementation of 1 g of EPA and DHA, regardless of fish consumption, was associated with a significant decrease in the PTX3 plasma concentration, in contrast with other inflammatory parameters [24]. These observations may confirm suggestions that the quantity of omega-3 acids in the Mediterranean diet is essential for secondary cardiovascular prevention.

In the study discussed here, no effect of supplementation of omega-3 acids on the remaining investigated parameters was found. Analysis of the obtained results suggests that this may be largely due to the lack of control over introduction of non-pharmacological secondary prevention (i.e. smoking cessation, adherence to the principles of Mediterranean diet, and adequate exercise). Studies suggested that introduction of those interventions reduced the risk of acute cardiovascular events or all-cause mortality [25]; however, the patient must be disciplined and strictly adhere to the doctor's instructions. Based on numerous studies, a trend towards decreased adherence with time of treatment may be observed. Such a trend was also observed in this study with respect to dietary modifications. In both investigated groups, after three months of supplementation, overall consumption of energy and saturated fatty acids was significantly higher ($p < 0.05$) and exceeded in both groups not only the value recommended for patients with cardiovascular diseases (7% of energy), but also the value recommended by World Health Organisation for healthy individuals (10% of energy). The consumption of polyunsaturated fatty acids, including omega-3 acids, decreased. Those changes were not statistically significant; nevertheless, they resulted in marked worsening of the PUFA/SAFA ratio, which in the investigated group decreased below 0.3 (with supplementation), while the recommended value is over 1. The results of 24-h interview were confirmed by those obtained using the food frequency questionnaire; meat and processed meat products as well as butter were consumed more often consumed ($p < 0.05$), while fish, vegetables, and fruit were consumed less frequently. Due to such large abnormalities in the proportion of fatty acids in the diet, the addition of just 1 g of omega-3 acids is not sufficient to improve this proportion to values characteristic for the Mediterranean diet. Worse adherence to the principles of the Mediterranean diet may be due to the lack of dietetic care after hospitalisation due to MI, resulting from a general lack of dietetic care in the Polish healthcare system.

Limitations of the study

The small number of patients participating in the study and short follow-up time might have affected the power of statistical analysis of the results.

CONCLUSIONS

In summary, it may be stated that supplementation of omega-3 acids in a dose of 1 g per day had no effect on lipid or inflammatory parameters, with the exception of PTX3. Probably, the potential benefits associated with supplementation were nullified by a highly atherogenic diet. Apparently, supplementation of omega-3 acids without simultaneous dietary education and nutrition control does not bring the expected effect. Further research involving a larger group of patients is needed to better understand the relationship between patient's diet and the effectiveness of omega-3 supplementation.

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Suplementacja kwasami omega-3 a modyfikacja markerów stanu zapalnego u pacjentów po zawale serca w świetle ich sposobu żywienia: badanie wstępne

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Streszczenie

Wstęp: Istotną rolę w rozwoju niewydolności serca (HF) odgrywa aktywacja neuroendokrynna, aktywacja cytokin prozapalnych i płytek krwi oraz dysfunkcja śródbłonka.

Cel: Celem pracy była wstępna ocena wpływu suplementacji kwasami EPA i DHA w dawce 1 g dziennie na wybrane markery stanu zapalnego i aktywacji płytkowej u pacjentów z HF po świeżo przebytym zawale serca, w świetle ich sposobu żywienia.

Metody: Badanie przeprowadzono w sposób randomizowany, metodą podwójnie ślepej próby, u 30 pacjentów z pozawałową HF. Jedna grupa otrzymywała preparat zawierający 1 g kwasów omega-3, druga grupa — olej kukurydziany w dawce 1 g dziennie przez 12 tygodni. Na początku badania i w 12. tygodniu pobrano na czczo krew żylną w celu oznaczenia następujących parametrów: NT-proBNP, fibrynogen, INR, klirens kreatyniny, lipidogram, hsCRP, troponina, glukoza, transaminazy, GGTP, MCP-1, pentraksyna-3, CD-40. Aby ocenić sposób żywienia i wielkość spożycia kwasów omega-3 z dietą, zastosowano 24-godzinny wywiad żywieniowy oraz kwestionariusz częstości spożycia kwasów omega-3 (FFQ) z modyfikacją Blocka.

Wyniki: Suplementacja kwasami omega-3 na poziomie 1 g dziennie nie przynosi efektów w postaci wpływu na parametry lipidowe i parametry stanu zapalnego, z wyjątkiem pentraksyny-3. W obu grupach po 3 miesiącach suplementacji spożycie energii i nasyconych kwasów tłuszczowych było istotnie większe wyjściowo ($p < 0,05$).

Wnioski: Potencjalne korzyści związane z suplementacją są prawdopodobnie niwelowane przez dietę o dużym stopniu aterogenności. Wydaje się więc, że suplementacja kwasami omega-3 w dawce 1 g dziennie bez jednoczesnej edukacji żywieniowej i kontroli spożycia nie przyniesie oczekiwanych efektów. Konieczne są dalsze badania obejmujące większe grupy pacjentów w celu lepszej oceny wpływu sposobu żywienia na efektywność suplementacji kwasami omega-3.

Słowa kluczowe: antygen różnicowania komórkowego 40 (CD-40), kwas dokozaheksaenowy (DHA), kwas eikozapentaenowy (EPA), markery stanu zapalnego, zawał serca, żywienie

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