ORIGINAL ARTICLE

Dietary intake of omega fatty acids and polyphenols and its relationship with the levels of inflammatory markers in men with chronic coronary syndrome after percutaneous coronary intervention

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C-reactive protein, inflammation, omega-3 fatty acids, omega-6 fatty acids, percutaneous coronary intervention, platelet-to-lymphocyte ratio

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ABSTRACT

BACKGROUND The platelet-to-lymphocyte ratio (PLR), neutrophil-to-lymphocyte ratio (NLR), and C-reactive protein (CRP) are useful in assessing inflammation in patients after percutaneous coronary intervention (PCI). The PLR and NLR are also independent predictors of cardiovascular mortality. Moreover, higher CRP levels increase the risk of long-term mortality in patients undergoing PCI.

AIMS We aimed to investigate the relationship between the dietary intake of omega-3 and omega-6 fatty acids and plant polyphenols and the levels of inflammatory markers in patients after PCI.

METHODS In this retrospective study, we used the validated Food Frequency Questionnaire and Aliant software to estimate the dietary intake of polyphenols and omega-3 fatty acids as well as the ratio of omega-6 to omega-3 fatty acids in patients after PCI. A total of 105 patients were divided into subgroups based on high or low dietary polyphenol intake, omega-3 fatty acid intake, and omega-6 / omega-3 fatty acid ratio. Data on complete blood count were obtained from the hospital laboratory.

RESULTS Patients with high omega-3 fatty acid intake (>4.18 g/d) had a lower PLR (P <0.03). Interestingly, only a low omega-6 / omega-3 ratio (below the cutoff value of 4:1) was associated with reduced CRP levels (P <0.008). There were no associations between dietary parameters and the NLR or between polyphenols and reduced levels of inflammatory markers.

CONCLUSIONS Anti-inflammatory effects of a diet should be assessed not only based on a high intake of omega-3 fatty acids but also balanced omega-6 / omega-3 ratio, which reduces PLR and CRP levels in patients with cardiovascular disease.

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INTRODUCTION Cardiovascular disease (CVD) is the leading cause of death worldwide, accounting for 17.9 million deaths every year. The inflammatory pathogenesis of atherosclerosis is well known.[1] A number of simple inflammatory markers such as C-reactive protein (CRP), platelet-to-lymphocyte ratio (PLR), and neutrophil-to-lymphocyte ratio (NLR) can be used to assess the anti-inflammatory effects of a diet. The PLR is a marker of inflammatory process and can be easily calculated from complete blood count.[2] It has been established as an independent prognostic factor for CVD.[3] Furthermore, it can be used as a predictor of acute and long-term death after percutaneous coronary intervention (PCI).[4] Similarly to the PLR, the NLR can be calculated based on complete blood count. It has been reported to have a prognostic value in CVD and is regarded as an independent predictor of cardiovascular death and long-term mortality in patients after PCI.[5] C-reactive protein is an acute-phase protein produced by the liver

WHAT'S NEW?

The platelet-to-lymphocyte ratio (PLR) is a novel inflammatory marker that has recently gained popularity, especially in the prediction of cardiac diseases and acute coronary syndrome. To the best of our knowledge, there is no information in the available literature on the use of both C-reactive protein (CRP) concentrations and PLR to monitor the anti-inflammatory effects of a diet. We found that consuming 4.2 g of omega-3 fatty acids per day with the concurrent omega-6 / omega-3 ratio of 4:1 or lower diminished the influence of inflammatory factors. Undoubtedly, CRP is used for monitoring as a single measure, but in our opinion, PLR could be applied as an additional diagnostic marker, which could facilitate the monitoring of anti-inflammatory effects of a diet. The cost-effectiveness of this approach would be an obvious advantage. Interestingly, although the simultaneous use of CRP and cellular markers for the assessment of inflammation is recommended in the literature, it does not directly refer to the monitoring of anti-inflammatory effects of a diet. Therefore, using both CRP and PLR is a novel diagnostic approach that follows recent trends.

in response to proinflammatory cytokines. According to the literature, the CRP level exceeding 3 mg/l increases the risk of CVD.[6] The CRP value independently predicts adverse cardiovascular events, including ischemic stroke, myocardial infarction, and sudden cardiac death.[7]

An anti-inflammatory diet should include a low or medium glycemic load, a low ratio of omega-6 to omega-3 fatty acids (at least below 5:1), a high concentration of omega-3 fatty acids, a high polyphenol intake, and individually adjusted caloric intake. Moreover, a high consumption of various vegetables and fruits as well as a proper balance between plant and animal proteins are recommended.[8,9] Polyphenols are considered beneficial in CVD due to their antioxidant and antithrombotic properties.[10] A high dietary polyphenol intake is correlated with reduced all-cause mortality and lower incidence of cardiovascular events. However, the mechanisms underlying this protective action are not fully understood.[11] Among potential cardioprotective effects of polyphenols, the following are postulated most frequently: inhibition of low-density lipoprotein oxidation, antioxidant action, improvement of endothelial function, and reduction of platelet aggregation.[12] Besides, antiplatelet and anti-inflammatory activity of plant polyphenols is well documented.[13,14] Numerous publications have reported that plant polyphenols reduce the CRP level in inflammatory diseases.[2,15]

Omega-3 fatty acids are known for their protective role in CVD.[16-18] In general, omega-6 fatty acids are considered proinflammatory, whereas omega-3 fatty acids—anti-inflammatory, mostly because they inhibit the production of arachidonic acid. The risk of several inflammatory diseases, including CVD, increases when the ratio of omega-6 to omega-3 fatty acids exceeds 5:1.[9] The omega-6 / omega-3 ratio is a more accurate parameter to assess the anti-inflammatory effect on the CRP level than the estimation of an omega-3 fatty acid intake alone.[19]

We hypothesized that a low ratio of omega-6 to omega-3 fatty acids as well as a high intake of omega-3 fatty acids and polyphenols can be beneficial for patients after PCI by reducing inflammation.

METHODS

Study design In this retrospective study, data were collected from June 2017 to December 2018 in the Intensive Cardiac Therapy Clinic (Medical University of Lodz, Łódź, Poland).

Participants We enrolled 105 men (mean [SD] age, 64.9 [8.6] years) with chronic coronary syndrome who were treated with multistage PCI. Patients were eligible for the study if they were 50 to 75 years old and were scheduled for PCI in the Intensive Cardiac Therapy Clinic. Patients were taking acetylsalicylic acid (75 mg/d) and clopidogrel (75 mg/d) or ticagrelor (90 mg twice daily). All patients were taking statins: rosuvastatin (20–40 mg/d) or atorvastatin (40–80 mg/d).

The exclusion criteria were as follows: history of intracranial bleeding, diabetes, stroke, renal dysfunction (estimated glomerular filtration rate <60 ml/min/1.73 m^2), primary and metastatic brain tumors, history of head trauma, as well as a major surgery or severe trauma within the past 6 weeks. The additional exclusion criterion was active (acute) inflammation defined as the CRP level exceeding 10 mg/l. Patients were advised not to use nonsteroidal anti-inflammatory drugs. The dietary exclusion criteria included an alternative diet (eg, a rigorous low-calorie diet, a vegetarian diet, or an elimination diet) and a daily caloric intake below 1000 kcal or above 5000 kcal.

For statistical analysis, patients were divided into groups based on polyphenol intake, omega-3 fatty acid intake, and the ratio of omega-6 to omega-3 fatty acids. The characteristics of the study group are presented in TABLE 1.

Laboratory tests Data on complete blood count and the CRP concentration were obtained from the hospital laboratory. The PLR and NLR values were calculated based on complete blood count. Cell blood counts were determined using the Sysmex XE-2100 analyzer (Sysmex Corporation, Kobe, Kansai, Japan). The CRP level was measured by a Tina-quant C-Reactive Protein Gen.3 immunoturbidimetric assay using the Modular Analytics EVO Cobas 6000 analyzer (Roche, Basel, Switzerland).

Dietary questionnaire and the estimation of fatty acid intake To estimate the nutritional value of a diet, including the content of phenolic compounds in vegetables, the previously validated Food Frequency Questionnaire (FFQ)

TABLE 1 Baseline characteristics of patients after percutaneous coronary intervention (n = 105)

Variable	Value
Age, y	64.9 (8.6)
Male sex, n (%)	105 (100)
BMI, kg/m^2	28 (4.8)
Hypertension, n (%)	94 (89.5)
Stable CAD, n (%)	105 (100)
Previous MI, n (%)	83 (79)
Diabetes, n (%)	0
Glucose, mmol/l	5.95 (0.84)
Dyslipidemia, n (%)	99 (94.3)
Total cholesterol, mmol/l	4.35 (1.17)
HDL cholesterol, mmol/l	1.02 (0.23)
Triglycerides, mmol/l	1.51 (0.85)
Physical activity (any level), n (%)	54 (51.4)
Family history of CVD, n (%)	69 (65.7)
Current smoking, n (%)	34 (32.4)
Aspirin, n (%)	105 (100)
Clopidogrel, n (%)	89 (84.8)
Ticagrelor, n (%)	16 (15.2)
Rosuvastatin, n (%)	59 (56.2)
Atorvastatin, n (%)	44 (46.2)
β-Blockers, n (%)	96 (91.1)
ACEIs, n (%)	87 (82.9)
Platelets, ×10^9/l	202 (61)
Lymphocytes, ×10^9/l	1.81 (0.57)
Neutrophils, ×10^3/l, median (IQR)	3.9 (3.05–4.7)
WBC, ×10^9/l	6.53 (1.74)
CRP, mg/l, median (IQR)	1.7 (1.1–3.73)
NLR, median (IQR)	2.08 (1.72–2.81)
PLR	120.1 (44.7)
MPVLR	5.63 (2.05)

Data are presented as mean (SD) unless otherwise indicated.

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; BMI, body mass index; CAD, coronary artery disease; CRP, C-reactive protein; CVD, cardiovascular disease; HDL, high-density lipoprotein; IQR, interquartile range; MI, myocardial infarction; MPVLR, mean platelet volume-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; WBC, white blood cell

was used. Based on the FFQ, a mean annual consumption of polyphenols and omega-3 fatty acids was estimated. The data were entered in a self-developed worksheet to estimate the content of the phenolic compounds. The worksheet was based on data from Phenol-Explorer, a comprehensive database that contains information about the quantity of 501 phenolic compounds (classified in 6 classes and 31 subclasses) in 459 food products. The worksheet included the following classes: a total quantity of vegetable phenolic compounds, flavonoids (alkylphenols, flavones, flavanols, catechins, procyanidins, anthocyanins, theaflavins, dihydrochalcones, isoflavonoids), flavan-3-ols, phenolic acids (hydroxybenzoic acid, hydroxycinnamic acid), stilbenes, and lignans. The data were entered in the Aliant software (Cambridge Diagnostics, Warsaw, Poland) to estimate the intake of omega-3 fatty acids and the ratio of omega-6 to omega-3 fatty acids. Patients were also asked about their intake of dietary supplements. The detailed daily dietary intake of various fatty acids and polyphenols in the whole PCI group is shown in TABLE 2.

Statistical analysis Normally distributed variables were presented as mean (SD), and the parameters with departures from normality (assessed by the Shapiro–Wilk test), as median and interquartile range. Variables that showed distribution other than normal were analyzed using the 2-tailed Mann–Whitney test. For categorical data, the Pearson χ^2 test with Yates correction (if necessary) was used. The Cohen κ coefficient was calculated as a measure of agreement. A κ value of less than 0.4 indicated poor to fair agreement; 0.41 to 0.60, moderate; 0.61 to 0.80, good; and 0.81 to 1, very good agreement. The Spearman rank correlation analysis was performed to assess the correlation between the studied parameters. For all the analyses, a P value of less than 0.05 was considered significant.

Ethics All patients gave informed consent to participate in the study. The study protocol was approved by the Committee for Medical Ethics (RNN/24/17/KE).

RESULTS In our study, patients after PCI (n = 105) were divided into subgroups with: 1) a low or high dietary intake of omega-3 fatty acids (the cutoff value was the median intake in the whole group, 4.2 g/d); 2) a low or high ratio of omega-6 to omega-3 fatty acids in diet (the cutoff value was the median ratio in the whole group, 4:1); and 3) a low or high dietary intake of plant polyphenols (the cutoff value was the median intake in the whole group, 1780 mg/d). All cutoff values are presented in detail in TABLE 3.

The subgroups were compared with respect to the levels of the inflammatory markers: CRP, PLR, and NLR. In the subgroup with a high dietary intake of omega-3 fatty acids, the PLR was significantly lower than in the low-intake group (mean [SD], 111.4 [40.2] vs 127.8 [41.6]; P = 0.03; FIGURE 1). The CRP and NLR levels did not differ between both subgroups. For the ratio of omega-6 to omega-3 fatty acids, the subgroup with a low ratio (<4:1) had a reduced CRP level compared with the high-ratio group

TABLE 2 Estimated daily dietary intake of fatty acids and polyphenols in patients after percutaneous coronary intervention (n = 105)

Variable	Dietary intake
Polyphenols, mg	1756 (1437–2331)
Flavan-3-ols, mg	508 (364–632)
Flavonoids, mg	854 (643–1140)
Phenolic acids, mg	419 (274–587)
Stilbenes, mg	0.13 (0.05–0.87)
Lignans, mg	20.97 (14.35–30.29)
Saturated fatty acids, g	33.48 (23.97–44.08)
MUFAs, g	44.97 (32.19–58.61)
PUFAs, g	21.53 (15.46–28.55)
Cholesterol, mg	332 (274–451)
EPA + DHA, g	0.73 (0.41–1.2)
Omega-3 fatty acids, g	4.15 (2.96–6.05)
Omega-6 fatty acids, g	16.60 (12.30–21.76)
Omega-6 / omega-3 ratio	3.81 (3.02–5.11)

Data are presented as median (Q1–Q3).

Abbreviations: DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids, Q1, lower quartile; Q2, upper quartile

TABLE 3 Cutoff points for the study subgroups

Dietary subgroup	Cutoff value	Median	Q1	Q3
Low omega-6 / omega-3 ratio	4:1	3:1	2.3:1	3.4:1
High omega-6 / omega-3 ratio		5.3:1	4.8:1	6.5:1
Low omega-3 fatty acid intake, g	4.2	3.0	2.1	3.7
High omega-3 fatty acid intake, g		6.1	5.3	7.8
Low polyphenol intake, mg	1780	1449	1079	1583
High polyphenol intake, mg		2369	1937	2951

Abbreviations: see TABLE 2

TABLE 4 Agreement between the levels of inflammatory markers and dietary omega-3 fatty acid intake in the study group

Dietary parameter vs inflammatory marker	Agreement, %	Cohen κ	95% CI
Omega-6/omega-3 fatty acid ratio vs CRP	69.3	0.39	0.17–0.59
Omega-3 fatty acid intake vs PLR	61.4	0.28	0.02–0.44

Abbreviations: see TABLE 1

(mean [SD], 1.83 [1.39] mg/l vs 2.71 [1.97] mg/l; $P = 0.007$; FIGURE 2). However, the PLR and NLR values were similar in both subgroups. Finally, no differences were found in the levels of inflammatory markers between the subgroups with a low and high dietary intake of plant polyphenols (Supplementary material, *Figure S1*).

We investigated an association between variables in the whole study group using 2 methods: agreement (see Statistical analysis) and Spearman rank correlation analysis. No association (either agreement or correlation) was found between the CRP, NLR, and PLR values and a total polyphenol intake. However, we found an association (both agreement and correlation) between the ratio of omega-6 to omega-3 fatty acids and the CRP level as well as between the omega-3 intake and the PLR value. The agreement between results was assessed using the Cohen κ test (TABLE 4). The Spearman correlation coefficient was 0.331 (95% CI, 0.128–0.503; $P < 0.001$) for the CRP level and the ratio of omega-6 to omega-3 fatty acids. For the PLR and omega-3 fatty acid intake, the correlation coefficient was –0.243 (95% CI, –0.4264 to –0.0304; $P = 0.03$). There was no correlation between the CRP and PLR values as well as between the CRP and NLR, but a correlation was noted between the PLR and NLR (correlation coefficient, 0.534; 95% CI, 0.341–0.711; $P < 0.001$).

DISCUSSION

Diet is considered one of the crucial factors in the pathogenesis and progression of both atherosclerosis and inflammation. It is believed that an anti-inflammatory diet should be rich in nutrients, such as omega-3 fatty acids, antioxidants (eg, vitamins C and E), fruits, vegetables, and flavonoids.[20] Therefore, it seems reasonable to monitor inflammatory markers and evaluate the anti-inflammatory potential of a diet in patients with CVD. Currently, a number of simple inflammatory markers are available, such as the CRP (a standard diagnostic marker detected in plasma) as well as the PLR and NLR (markers easily calculated from complete blood count).[2,21] In this study, we attempted to verify whether the total dietary intake of omega-3 fatty acids, the ratio of omega-6 to omega-3 fatty acids, and plant polyphenol intake affect the CRP, PLR, and NLR levels in patients after PCI. We found that the PLR was significantly reduced in the subgroup of patients after PCI who had a high intake of omega-3 fatty acids; however, the NLR and CRP values were not reduced in this group. Interestingly, the PLR is regarded as an independent prognostic factor for CVD [3] as well as a predictor of acute and long-term death after PCI.[4] An elevated PLR (>150) was found to be a predictor of all-cause mortality and cardiovascular events.[22]

Our results are in line with a general consensus that dietary nutrients such as omega-3 fatty acids found in fish, plant polyphenols, as well as antioxidants like vitamin E and flavonoids are associated with less severe inflammation.[23,24] Regarding the lack of significance for the CRP level, numerous clinical trials assessed whether supplemental eicosapentaenoic and / or docosahexaenoic acid can reduce the plasma CRP level,

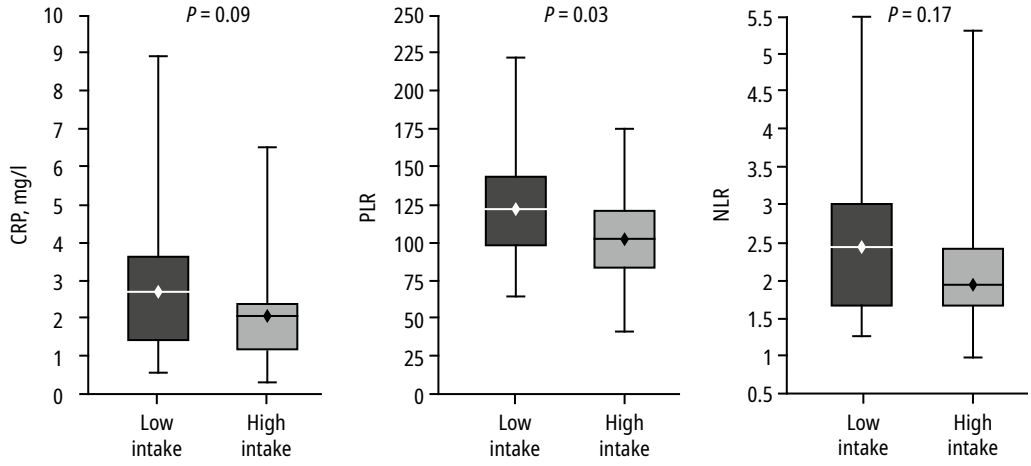

FIGURE 1 Effects of dietary omega-3 fatty acid intake on the C-reactive protein (CRP) level, platelet-to-lymphocyte ratio (PLR), and neutrophil-to-lymphocyte ratio (NLR) in low- and high-intake groups. Data are shown as median (interquartile range, minimum and maximum values).

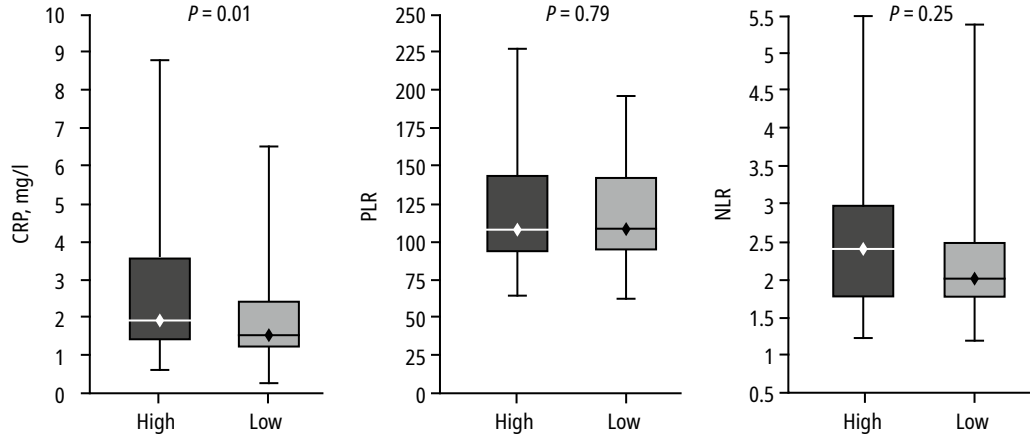

FIGURE 2 Effects of the dietary ratio of omega-6 to omega-3 fatty acids on the C-reactive protein (CRP) level, platelet-to--lymphocyte ratio (PLR), and neutrophil-to-lymphocyte ratio (NLR) in the groups with a high and low ratio. Data are shown as median (interquartile range, minimum and maximum values).

but it was not typically a primary endpoint.[25] It is important to note that the impact of omega-3 supplementation differs in patients at risk of CVD. Furthermore, a number of studies reported that supplementation of omega-3 fatty acids at a dose of 0.6 to 1.7 g/d did not have a significant effect on the CRP level in patients with CVD.[26,27] In a study by Samavat et al,[28] where the supplementation of omega-3 fatty acids was very high (4 g/d for 8 weeks), no major difference in the high-sensitivity CRP level was found between the interventional and placebo groups of patients with CVD. Besides, in obese and severely obese adults as well as in those with hypertension, diabetes, or hypertriglyceridemia, the supplementation of omega-3 fatty acids at a dose of 2.6 to 4.2 g/d did not influence the CRP level, whereas in adults with dyslipidemia, a decrease in the CRP level was observed.[29] Omega-3 fatty acids can also improve the platelet response to antiplatelet therapy. Some reports showed that the omega-3 fatty acid supplementation of 1 g/d for 1 month significantly reduced the $P2Y_{12}$ reactivity index when used in addition to dual antiplatelet therapy.[30,31] Since poor platelet response to clopidogrel, one of the $P2Y_{12}$ inhibitors, is associated with a prolonged increase in the CRP level after PCI, supplementation with omega-3 fatty acids, which improve the platelet response to clopidogrel and decrease the risk of a prolonged CRP increase, can be beneficial in patients after PCI.[32]

So far, no studies have investigated the effect of omega-3 fatty acids or plant polyphenols on the NLR level. Therefore, the NLR cannot be currently used to assess the anti-inflammatory impact of a diet. Interestingly, a recent study has demonstrated that diet modification affects the NLR value in patients after surgery.[21]

Since not only a total dietary intake of omega-3 fatty acids but also the ratio of omega-6 to omega-3 essential fatty acids is important,[33] we also

looked at the inflammatory markers in the subgroups with a low and high ratio of omega-6 to omega-3 fatty acids. We demonstrated that the subgroup with a low ratio (<4:1) had a significantly reduced CPR level; however, the PLR and NLR values did not differ between subgroups. Generally, excessive amounts of omega-6 polyunsaturated fatty acids (PUFAs) and a high ratio of omega-6 to omega-3 fatty acids, typically found in Western diets, promote the pathogenesis of numerous diseases, including cardiovascular disorders, cancer, as well as inflammatory and autoimmune conditions, whereas increased omega-3 PUFA levels (a low omega-6/omega-3 ratio) have a suppressive effect. In the secondary prevention of CVD, the ratio of 4:1 was associated with a 70% reduction in total mortality.[34] Moreover, a similar correlation was shown for fatty acid composition in plasma. Patients with myocardial infarction had a significantly higher omega-6/omega-3 ratio than those at cardiovascular risk.[35]

Ferrucci et al[36] studied the relationship between plasma PUFAs and circulating inflammatory markers in 1123 individuals aged 20 to 98 years in a community-based sample. The total amount of omega-3 fatty acids was independently associated with lower levels of proinflammatory markers (interleukin [IL] 6, IL-1 receptor antagonist, tumor necrosis factor α, and CRP) and higher levels of anti-inflammatory markers (soluble IL-6 receptor, IL-10, and transforming growth factor α), independent of confounders. The ratio of omega-6 to omega-3 fatty acids showed a strong negative correlation with the level of IL-10.[36]

The beneficial impact of polyphenols in CVD is multifactorial, but their most important role is the reduction of chronic and acute inflammation. The possible mechanisms of anti-inflammatory effects of polyphenols involve antioxidant activity, modification of signaling cascades and transcriptional networks, as well as reduction of immune cell adhesion and endothelial dysfunction.[37] In a large prospective cohort study, involving 18 countries, dietary fruit intake was associated with a lower risk of noncardiovascular, cardiovascular, and all-cause mortality, while legume intake was inversely correlated with all-cause and noncardiovascular mortality. Consumption of 3 to 4 portions (375–500 g/d) of raw vegetables daily appears to be most beneficial for reducing both noncardiovascular and all-cause mortality.[38]

In our study, we did not observe any effect of plant polyphenols in diet on the levels of any inflammatory markers studied. Similarly, no significant effect of polyphenols on the reduction in CRP levels has been reported in the available literature. There is no evidence for a short-term beneficial effect of polyphenol intervention on microcutaneous vascular response and pulse wave velocity on measurable risk factors in overweight or obese patients. Besides, no significant differences were seen in the plasma levels of leptin, apolipoproteins, cystatin C, insulin, adiponectin, CRP, intercellular adhesion molecule 1, E-selectin, or tissue plasminogen activator, but the IL-6 level increased in active versus placebo recipients (0.32 pg/ml vs 0.18 pg/ml; $P = 0.01$).[39] Quercetin was reported to reduce CRP expression in mice, although this finding has not been verified in clinical trials.[40] Using the PLR for monitoring anti-inflammatory effects of a diet is a novel approach, but it is also in line with a recent systematic review by Kurtul and Ornek including 111 studies,[41] which provided evidence for the association of the PLR with CVD and its possible use as a prognostic marker of CVD.

Study limitations The FFQ is a common tool for a retrospective assessment of the nutritional value of a diet. Nevertheless, it has some limitations: the frequency and quantity of product consumption in the past year is self-reported by patients, which requires the ability to perform complex cognitive tasks that involve memory and mathematical thinking (averaging). Furthermore, a significant limitation of the study is its retrospective design and the fact that no objective indicators (ie, biomarkers: omega-3 and omega-6 PUFAs in blood/serum/tissue and polyphenol metabolites) were used to verify the actual dietary intake of omega-3 and omega-6 PUFAs and polyphenols in study participants. Moreover, the group size was relatively small and research on a larger group of patients would provide more reliable results.

Conclusions In summary, not only a total intake of omega-3 PUFAs but also a balanced omega-6/omega-3 ratio (below 4:1) should be maintained in the anti-inflammatory diet in cardiovascular patients. In addition, the use of a multiparametric approach and not only the CRP level but also indices derived from complete blood count, such as the PLR, is recommended for monitoring the anti-inflammatory effect of a diet.

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

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CONFLICT OF INTEREST None declared.

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