

Does heart failure determine the nutrition of patients?

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Abstract

Background: Studies of the diet of heart failure (HF) patients, available in the literature, indicate that this diet is highly inadequate in light of the existing recommendations.

Aim: To assess the nutrition of middle-aged patients with HF compared to a control group representing the general population without HF.

Methods: The study group comprised 70 patients with diagnosed HF, below 65 years, with NYHA class I/II/III. For the control group, we selected members of the general population with identical sex, age, body mass index and cigarette smoking parameters as the tested persons. Diet was evaluated on the basis of a dietary questionnaire and a 24-h dietary recall.

Results: The energy/ideal body weight ratio was significantly lower in the study group than in the control group ($p < 0.005$). Patients with HF had lower % kcal from fat, SAFA, MUFA ($p < 0.05$), omega-3 ($p < 0.05$), folic acid ($p < 0.001$) and dietary fibre ($p < 0.01$) consumption compared to the control group. No intergroup differences were found in the consumption of vitamins B6, C, A or E. The lowest energy, dietary fibre and vitamins intake (except vitamin A) was found in patients in NYHA class III.

Conclusions: The diet of patients with HF seems to differ from the diet of persons without HF. These differences are manifested in a lower energy content of diet, lower intake of total fat, omega-3 fatty acids, folic acid and dietary fibre. Most patients with HF have ischaemic aetiology, and therefore should apply the dietary recommendations for patients with dyslipidaemia and high cardiovascular risk. However, their diet for certain parameters was inconsistent with ESC/EAS guidelines (high intake of saturated fatty acids, low intake of dietary fibre).

Key words: nutrition, heart failure, NYHA class, general population

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INTRODUCTION

Promoting healthy nutritional habits plays an important role in the treatment of patients with cardiac disorders, including heart failure (HF). Unfortunately, there have been no detailed nutritional requirements available so far which would ensure appropriate intake of nutrients in this patient group. However, it should be noted that attempts to develop dietetic recommendations for patients at risk of HF have recently been made, but not for patients with this disease actually diagnosed [1, 2]. Studies of the diet of HF patients, available in the literature, indicate that this diet is highly inadequate in light of the existing recommendations. This is particularly noticeable in groups of

patients with advanced stages of HF, i.e. of New York Heart Association (NYHA) class III/IV — an insufficient energy intake is found, especially in the elderly. Furthermore, a low intake of vitamins and minerals in diets of this patient group, which often does not exceed 50% of the recommended delivery, must raise concern. The study by Lemon et al. [3], found calcium, magnesium, omega-3 acid and dietary fibre intake consistent with the requirements in only 13%, 10%, 2% and 4% of the HF study subjects, respectively. The authors concluded that the quality of nutrition in that patient group was very bad, and that more effective dietary interventions were necessary to improve this condition [3]. Similar observations were made

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by Catapano et al. [4] in patients with HF above 60 years of age and by Arcand et al. [5] in patients with stable HF. Keith et al. [6] confirmed in their study a deficit of B vitamins in the blood serum in patients with HF, which was due to a considerable deficiency of the foods constituting the dietary source of these vitamins in their everyday diet. It could be that one of the causes of this status is unawareness of the potential benefits of therapy, and of the prognosis of further risk arising from inappropriate nutrition. Heo et al. [7] indicated that there were no educational strategies available for this patient group which would allow increasing their knowledge about healthy nutrition and the importance of such nutrition as an adjunctive element in therapy and secondary prophylaxis, to improve the comfort of patients' everyday life.

Our study objective was to assess the nutrition of middle-aged (below 65 years of age) patients with HF, broken down by NYHA class, compared to a control group representing the general population without HF, selected on the basis of age and anthropometric criteria.

METHODS

Patients (both men and women) with diagnosed HF of NYHA class I, II or III, below 65 years of age, treated on an outpatient basis in cardiology clinics were eligible for inclusion in the

study group. HF was diagnosed on the basis of an ECG, an echocardiographic examination and an exercise test. In 80% of the cases, this was HF with ischaemic aetiology developed as a consequence of myocardial infarction. Patients hospitalised within the two months before study initiation, patients with HF classified as NYHA class IV, and patients with cancer, autoimmune disorders and morbid obesity (body mass index > 39.9) were excluded from the study. For inclusion in the control group, we selected members of the general population with identical sex, age, body mass index and cigarette smoking parameters as the tested persons. The exclusion criteria were HF, ischaemic heart disease or hypertension which had been treated for longer than three years, chronic liver and kidney disease. The clinical characteristics of both groups are presented in Table 1. The patients' nutrition was assessed with the use of a 24-h nutritional interview method. The energy and nutrient contents were calculated with the use of the Polish computer program Dietetyk 2 developed by the National Food and Nutrition Institute. The study omitted the amount of fluids consumed (patients do not have recommendations limiting the amount of fluid) and sodium intake (most of the respondents were men who were not able to assess the amount of salt used for food preparation). Nutrient contents were compared with the ESC/EAS guidelines for the management of dyslipidaemia

Table 1. Clinical characteristics of the study and control groups

	Study group (n = 70)	Control group (n = 70)
Age [years]	56.8 ± 6.2	56.5 ± 7.1
Sex: men	49 (70%)	49 (70%)
Education: primary/professional/secondary/higher	18/12/25/15 (24/18/35/22%)	20/19/22/9 (28/27/31/14%)
Employment status: economically active/retired/unemployed	16/54/0 (23/77/0%)	19/46/5 (27/66/7%)
Marital status (married/single)	60/10 (86/14%)	62/8 (89/11%)
Body mass index [kg/m ²]	29.4 ± 3.4	29.5 ± 6.2
Smokers	18 (25%)	18 (25%)
Post-myocardial infarction	56 (80%)	–
Left ventricular ejection fraction	38.7 ± 6.6	–
NYHA class I/II/III	20/35/15 (28/50/22%)	–
Diabetes mellitus	26 (37%)	–
Hypertension	56 (80%)	–
Treatment:		
Beta-blocker	66 (94%)	22 (31%)
ACE inhibitors	56 (80%)	36 (51%)
Statin	56 (80%)	–
NYHA class I/II/III	15 (75%)/30 (85%)/11 (73%)	–
Furosemide	28 (40%)	–
Fenofibrates	6 (8%)	–
Digoxin	6 (8,5%)	–
Acetylsalicylic acid	49 (70%)	–
Insulin	14 (20%)	–
Metformin	12 (17%)	–

[8] and human nutrition standards [9]. Blood for biochemical tests was drawn in a fasting state, a minimum of 12 h after the last meal, before nutritional history was taken. Laboratory tests included serum total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides and homocysteine. Triglycerides, TC and HDL-C were measured using traditional methods. LDL-C was calculated from the Friedewald formula. Homocysteine expressed in $\mu\text{mol/L}$ was measured by fluorescence polarisation immunoassay (Abbott IMX Analyzer; Abbott Park, IL, USA). All continuous variables were presented as arithmetic means \pm standard deviations and compared with the use of a test based on the t-Student distribution. Statistical analysis was performed with the use of the STATISTICA StatSoft Polska v. 9.0 package (StatSoft Inc., Tulsa, OK, USA). This study was approved by the Bioethics Committee of the National Food and Nutrition Institute in Warsaw.

RESULTS

Table 2 presents the contents of energy and selected nutrients in the diets of the study and control groups, on the basis of the 24-h nutritional history. Table 3 presents mean values of biochemical parameters of atherosclerosis risk assessment.

The results obtained indicated significant differences in the nutrition of patients with HF compared to representatives of the general population. A noticeable finding in the analysis of the diets of the study group and of the control group was a statistically significant difference in the energy content of the diet (1,645 kcal vs. 1,969 kcal, $p < 0.001$). Patients in NYHA class III had a much lower energy value of the diet than patients with less advanced disease. Similarly, the energy/ideal body weight ratio was significantly lower in the study group than in the control group ($p < 0.005$). When analysing energy sources, it should be stated that statistically significant differences were observed for protein and fat consumption but not for carbohydrate consumption. Patients with HF were characterised by a lower percentage of energy originating from fat (31.2%) compared to the control group (34.5%, $p < 0.05$). They also had a higher percentage of energy from protein (17.7% of total energy vs. 14.3%, $p < 0.0001$), although the total protein content was similar in both groups. Furthermore, methionine, an amino acid characteristic for animal protein, was assessed, and no differences were found between both groups. Consumption of carbohydrates as the main source of energy was at lower recommended levels in both groups (51%). No intergroup differences were found in the consumption of vitamins B6, C, A or E. This difference was statistically significant only in the case of folic acid (FA) ($p < 0.001$).

DISCUSSION

Our study demonstrates significant differences both between the nutrition of patients with HF and the nutrition of the general population, as well as between groups of patients

with HF selected on the basis of the severity of the disease. The low energy/ideal body weight ratio found, deviating from the guidelines (for ambulatory patients it should be 30 kcal/kg body weight), may indicate an increased risk of malnutrition in patients with a severe disease, which has been proven to constitute one of the causes of its rapid progression. In contrast to conventional cardiovascular disease (CVD) risk factors and overfeeding, which need from less than ten to more than ten years to cause a lethal effect, the effect of the nutrition-inflammation-cachexia complex is manifested rapidly, resulting in a decreasing short-term survival [10].

In accordance with the ESC/EAS and HF Society guidelines for the management of dyslipidaemia [8, 11] in patients with high risk CVD, it is recommended to obtain a LDL-C level below 100 mg/dL, and in patients with high risk CVD of below 70 mg/dL [8, 11]. In this study, 37% had LDL-C below 100 mg/dL and only 7% below 70 mg/dL; they were patients treated with statins at doses above 40 mg/day. Therefore, it seems that although statin use contributed to obtaining the TC and LDL levels significantly lower than in the general population, these results are still unsatisfactory, the cause of which may be the use of insufficient statin doses. It is also interesting that the atherogenicity ratio TC/HDL did not differ between both groups in view of the significantly lower HDL-C level in the test group ($p < 0.0001$). This seems to be of concern because studies indicate that high HDL levels in patients with HF are associated with lower mortality [12]. In fact, as was demonstrated in the Framingham Study, dyslipidaemia is associated with a risk of HF regardless of its association with myocardial infarction, which suggests that the effect on the lipid level may be a method of reducing HF risk. In fact, a high baseline non-HDL level was found to increase the HF risk by 29%, while a low HDL-C level is associated with a 40% higher risk of HF [13]. Because pharmacological treatment of the patients in the test group was based on the European standards, it seems that ineffectiveness of this treatment with respect to obtaining satisfactory levels of lipids should also be attributed to diet quality, and especially to the type of fats consumed.

As shown in the presented study, the percentage of energy from fat was lower in the study group and was consistent with the ESC/EAS recommendations ($< 35\%$ energy of diet). It is well established that a high (above 10% of energy) intake of saturated fatty acids causes an increase in the level of TC in the serum, an increase in the LDL fraction, an increase in the production of the very low-density lipoprotein fraction, and also contributes to increased blood coagulability, endothelial dysfunction, increased blood pressure or arrhythmia. Therefore, it may be of concern that the percentage of energy from saturated fatty acids in the diet of HF patients was more than 12%, which significantly exceeds the value recommended by the ESC/EAS ($< 7\%$ energy of diet) [8]. These results are definitely less positive than in other studies; for example, in the study by Arcand et al. [5], saturated fatty acids (SAFA)

Table 2. Energy and selected nutrient content in study group and control group diets

	Study group		Control group	P
	HF groups	HF mean		
Energy [kcal]	NYHA I: 1,639.6 ± 897.4 NYHA II: 1,712 ± 832.9 NYHA III: 1,535 ± 566	1,645.8 ± 796.6	1,969.8 ± 788.1	< 0.001
Energy/ideal body weight	NYHA I: 23.5 ± 13.9 NYHA II: 25.9 ± 11.4 NYHA III: 24.3 ± 13.8	24.9 ± 11.6	30.6 ± 12.4	< 0.005
Energy/actual body weight	NYHA I: 18.4 ± 10.9 NYHA II: 18.8 ± 8.0 NYHA III: 19.6 ± 11.6	18.6 ± 8.7	23.1 ± 9.8	< 0.005
% kcal from fat	NYHA I: 28.13 ± 10.4 NYHA II: 32.3 ± 6.37 NYHA III: 32.8 ± 11.3	31.2 ± 8.9	34.5 ± 8.0	< 0.05
% kcal from protein	NYHA I: 18.35 ± 4.6 NYHA II: 17.34 ± 6.3 NYHA III: 17.65 ± 5.2	17.7 ± 5.6	14.3 ± 3.6	< 0.0001
% kcal from carbohydrates	NYHA I: 53.52 ± 11.1 NYHA II: 50.36 ± 9.37 NYHA III: 49.5 ± 13.1	51.0 ± 10.7	51.1 ± 9.19	NS
SAFA [g] (% kcal from SAFA)	NYHA I: 22.9 ± 21.15 (11.8 ± 5.3) NYHA II: 23.9 ± 13.9 (12.6 ± 4.6) NYHA III: 20.9 ± 10.95 (12.4 ± 5.9)	22.8 ± 15.6 (12.7 ± 8.7)	27.9 ± 15.2 (12.7 ± 6.9)	< 0.05 NS
MUFA [g] (% kcal from MUFA)	NYHA I: 26.19 ± 24.68 (13.1 ± 5.2) NYHA II: 26.7 ± 14.7 (14.1 ± 13.7) NYHA III: 21.7 ± 9.9 (13.05 ± 5.1)	25.5 ± 7.0 (13.9 ± 9.4)	30.9 ± 17.8 (14.1 ± 8.12)	0.05 NS
PUFA [g] (% kcal from PUFA)	NYHA I: 12.15 ± 12.49 (6.6 ± 7.5) NYHA II: 11.53 ± 6.64 (6.1 ± 2.8) NYHA III: 11.46 ± 12.3 (5.8 ± 4.2)	11.7 ± 9.7 (6.4 ± 5.4)	11.9 ± 7.4 (5.4 ± 3.3)	NS NS
Omega-3 acids [g] (% kcal from omega-3)	NYHA I: 0.70 ± 0.245 (0.62 ± 0.36) NYHA II: 1.36 ± 1.043 (1.17 ± 0.89) NYHA III: 0.69 ± 0.44 (0.24 ± 0.008)	1.23 ± 0.91 (0.73 ± 0.529)	1.75 ± 1.159 (1.01 ± 0.807)	< 0.005 < 0.05
PUFA/SAFA ratio	NYHA I: 0.53 NYHA II: 0.48 NYHA III: 0.52	0.51	0.42	< 0.05
Methionine [mg]	NYHA I: 1,869.6 ± 1,314.9 NYHA II: 1,315.2 ± 902.3 NYHA III: 1,464.33 ± 792.5	1,555.5 ± 994.4	1,576.4 ± 702.1	NS
Cholesterol [mg]	NYHA I: 249 ± 277.4 NYHA II: 332 ± 549.8 NYHA III: 236 ± 169.15	207.2 ± 424.8	252.6 ± 139.2	NS
Vitamin B6 [mg]	NYHA I: 1.92 ± 1.48 NYHA II: 1.72 ± 0.72 NYHA III: 1.56 ± 0.97	1.74 ± 1.0	1.84 ± 0.7	NS
Folate [μg]	NYHA I: 188.9 ± 83 NYHA II: 191.82 ± 90 NYHA III: 134.17 ± 57	177.2 ± 84.9	240.1 ± 129.1	< 0.001

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Table 2. (cont.) Energy and selected nutrient content in study group and control group diets

	Study group		Control group	P
	HF groups	HF mean		
Vitamin A [μg]	NYHA I: 853.1 \pm 1,332.48 NYHA II: 1,068 \pm 2,018 NYHA III: 1,099.2 \pm 1,490	1,013.3 \pm 1,722.3	1,018.3 \pm 1484.4	NS
Vitamin E [mg]	NYHA I: 8.25 \pm 4.22 NYHA II: 9.77 \pm 5.7 NYHA III: 6.52 \pm 4.8	8.7 \pm 5.2	10.2 \pm 5.47	NS
Vitamin C [mg]	NYHA I: 100.6 \pm 107.68 NYHA II: 76.52 \pm 61.1 NYHA III: 56.9 \pm 45.88	79.4 \pm 75.8	81.2 \pm 60.2	NS
Dietary fibre [g]	NYHA I: 17.05 \pm 9.4 NYHA II: 17.07 \pm 9.01 NYHA III: 14.7 \pm 9.32	16.9 \pm 9.15	20.8 \pm 8.19	< 0.01

p value between study heart failure (HF) mean group and control group; SAFA — saturated fatty acids; MUFA — monounsaturated fatty acid; PUFA — polyunsaturated fatty acid

Table 3. Mean values of biochemical parameters in study and control groups

	Study group		Control group	P
	HF groups	HF mean		
Total cholesterol [mg/dL]	NYHA I: 172.8 \pm 43.4 NYHA II: 180.6 \pm 43.2 NYHA III: 210.2 \pm 41.3	180.4 \pm 44.5	212.1 \pm 39.1	< 0.001
HDL cholesterol [mg/dL]	NYHA I: 39.15 \pm 6.9 NYHA II: 44.2 \pm 9.37 NYHA III: 37.3 \pm 11.4	41.3 \pm 9.6	51.9 \pm 12.5	< 0.0001
LDL cholesterol [mg/dL]	NYHA I: 108 \pm 41.3 NYHA II: 114 \pm 44.4 NYHA III: 155 \pm 44.3	119.6 \pm 14.5	133.8 \pm 33.4	< 0.05
Triglycerides [mg/dL]	NYHA I: 181.2 \pm 155.3 NYHA II: 148.6 \pm 89.3 NYHA III: 155.7 \pm 163.1	144.9 \pm 101.1	138.6 \pm 82.7	NS
Homocysteine [mmol/L]	NYHA I: 12.4 \pm 3.1 NYHA II: 14.06 \pm 4.5 NYHA III: 13.25 \pm 3.6	13.9 \pm 4.05	10.6 \pm 3.2	< 0.005

p value between study heart failure (HF) mean group and control group

intake was 9.3% of dietary energy. Similarly as in the case of energy from fat, the lowest percentage of energy originating from SAFA was found in the group of patients at an early stage of the disease, which may be explained by the fact that at that stage, more emphasis is put on non-pharmacological treatment methods, including dietetic methods, whereas when the disease develops, treatment is focused mainly on pharmacological methods. The study found a significantly lower intake of omega-3 ($p < 0.005$) resulting in a lower

percentage of energy from omega-3 ($p < 0.05$). Most of the intake of omega-3 accounted for alpha-linoleic acid — only six patients in the study group ate fish and fish products on the day on which testing was performed. This is alarming because omega-3 fatty acids from fish, but not alpha-linoleic acid, has been shown to benefit CVD outcomes in primary and secondary prevention studies [14, 15].

An index of a diet which is correctly balanced with respect to fat content is the P:S ratio, i.e. polyunsaturated fatty acids

(PUFA) to SAFA ratio, which should be close to 1. In the presented study, the P:S ratio was negative both in the study group (0.51) as well as in the control group (0.42) ($p < 0.05$), with a similar amount of PUFA in both diets. In the diet of patients with HF, a higher content of dietary cholesterol was found (287 mg vs. 251 mg in the control group), not exceeding the ESC/EAS recommendations, that is 300 mg/day [8]. However, the highest intake of dietary cholesterol was observed in the NYHA class II group (332 ± 549.8 mg).

Another element of the diet which might have a negative effect on the lipid profile was dietary fibre content, which was significantly lower in the diet of patients with HF than in the control group (16.9 vs. 20.8 g; $p < 0.01$). The cause was insufficient ingestion of vegetables and products containing complex carbohydrates. Only 14% of HF patients met the valid recommendation of consuming 25 g/day of dietary fibre [8]. Unfortunately, most studies of diet quality of HF patients do not take into account the content of dietary fibre, focusing mainly on energy value and contents of macro- and micronutrients. Only Lemon et al. [3] estimated that dietary fibre ingestion by HF patients in the NHANES 1999–2006 study was very low and as little as 4% of the study population ingested the recommended quantities of fibre. Low consumption of fibre in the test group may be one of the causes of the negative lipid profile, despite using pharmacotherapy with statins and fibrates.

Vitamin content is of particular importance in nutrition quality assessment. As shown by the presented study, low delivery of FA raises particular concern. The mean folate content in the diet of HF patients was only 177 μg ; in the control group, it was 240 $\mu\text{g}/\text{day}$, and this was a statistically significant difference ($p < 0.001$). A comparison of these values with the recommended values means that it is only 44% of Recommended Dietary Allowances (RDA) in the study group and 60% of RDA in the control group. Such low consumption may have an effect on many biochemical processes in the body. The positive effect of FA on the homocysteine level is well known. In our study, the homocysteine level was found to be statistically significantly higher in the study group than in the control group (13.3 vs. 10.6 $\mu\text{mol}/\text{L}$, $p < 0.005$). Because the contents of methionine and vitamin B6 in both study groups were similar (1.7 mg in the test group vs. 1.8 mg in the control group), FA deficiency might have constituted one of the causes of hyperhomocysteinaemia. In the NYHA class I group, the dietary content of FA was the highest, and at the same time homocysteine level was the lowest. Many authors have suggested that the potential positive effect of FA on the vascular epithelium may also be exerted otherwise than through homocysteine [16, 17]. The beneficial effect of FA reducing the risk of cardiovascular disorders may result from its effect on nitrogen oxide activity, free radical reductions and coagulation mechanism. As has been shown in animal studies, a diet with a low content of FA contributes to an increased aggregation activity and increases the tissue factor activity in macrophages [18].

There are limited studies as to whether a healthy diet high in antioxidants, such as vitamin C and vitamin E, may help prevent HF [19]. In accordance with Polish RDA, the recommended intake of vitamin C is 75 mg in women and 90 mg in men and the recommended intake of vitamin E is 8 mg in women and 10 mg in man [9]. In the presented study, the mean intake of vitamin C in the study group (79 ± 75.8 mg) was lower than in the control group (81 ± 60.2 mg) but the differences were not statistically significant. The lowest intake was found in patients in the NYHA class III group ingesting 44% less vitamin C than NYHA class I patients. As to vitamin E, the mean intake in the study group was lower than in the control group (7.21 mg in women, 9.41 in men vs. 8.11 mg in women, 11.1 in men). Only 36% of women and 20% of men in the study group consumed the recommended amounts of vitamin E (in the control group, 37% of women and 30% of men). Considerable vitamin E deficiencies in the diet were observed also by Catapano et al. [4] and Arcand et al. [5] in almost all HF patients.

Therefore, it seems that HF patients are a group at particular risk of tocopherol deficit in the diet, which may contribute to their higher susceptibility to oxidative stress and to an imbalance between thromboxane and prostacyclin, which in turn may lead to predominance of the vasoconstrictive platelet aggregation-increasing action.

The only vitamin whose mean intake in the group was higher than recommended was vitamin A. The mean intake of vitamin A (as μg of retinol) in both groups (the study group 1,013 μg and the control group 1,018 μg) exceeded the RDA values (900 μg for men and 700 μg for women). However, very high inter-individual variations of intake of this vitamin were found, which was indicated by high standard deviation values. However, taking into account the RDA values for vitamin A, in 68% of patients in the HF group, its intake was lower than recommended. This is consistent with the study by Catapano et al. [4] who demonstrated that the group of HF patients meeting the RDA recommendations was only 21%. The finding of the highest vitamin A content in the diet of patients of NYHA class III (1,099 μg) is interesting; this is probably connected with a high delivery of animal products, which is also indicated by a high dietary content of SAFA in this patient group.

CONCLUSIONS

The diet of patients with HF seems to differ from the diet of persons without HF. These differences manifest in a lower energy content of diet, lower intake of total fat, omega-3 fatty acids, FA and dietary fibre. Most patients with HF have ischaemic aetiology, and therefore should apply dietary recommendations for patients with dyslipidaemia and high cardiovascular risk. However, their diet for certain parameters was inconsistent with ESC/EAS guidelines (high intake of SAFA, low intake of dietary fibre).

Conflict of interest: none declared

References

- Lennie TA. Nutritional recommendations for patients with heart failure. *J Cardiovasc Nurs*, 2006; 21: 261–268.
- Schocken DD, Benjamin EJ, Fonarow GC et al. Prevention of heart failure: a scientific statement from the American Heart Association Councils on Epidemiology and Prevention, Clinical Cardiology, Cardiovascular Nursing, and High Blood Pressure Research; Quality of Care and Outcomes Research Interdisciplinary Working Group; and Functional Genomics and Translational Biology Interdisciplinary Working Group. *Circulation*, 2008; 117: 2544–2565.
- Lemon SC, Olendzki B, Magner R et al. The dietary quality of persons with heart failure in NHANES 1999–2006. *J Gen Intern Med*, 2010; 25: 135–140.
- Catapano G, Pedone C, Nunziata E et al. Nutrient intake and serum cytokine pattern in elderly people with heart failure. *Eur J Heart Fail*, 2008; 10: 428–434.
- Arcand J, Floras V, Ahmed M et al. Nutritional inadequacies in patients with stable heart failure. *J Am Diet Assoc*, 2009; 109: 1909–1913.
- Keith ME, Walsh NA, Darling PB et al. B-vitamin deficiency in hospitalized patients with heart failure. *J Am Diet Assoc*, 2009; 109: 1406–1410.
- Heo S, Lennie TA, Moser DK et al. Heart failure patients' perceptions on nutrition and dietary adherence. *Eur J Cardiovasc Nurs*, 2009; 8: 323–328.
- Reiner Z, Catapano AL, De Backer G et al. ESC/EAS Guidelines for the management of dyslipidaemias: the Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). *Eur Heart J*, 2011; 32: 1769–1818.
- Jarosz M, Bulhak-Jachymczyk B. Human nutrition standards. Basic prevention of obesity and non-communicable diseases. PZWL, Warsaw 2011.
- Kalantar-Zadeh K, Abbott KC, Kronenberg F et al. Epidemiology of dialysis patients and heart failure patients; special review article for the 25th anniversary of the Seminars in Nephrology. *Semin Nephrol*, 2006; 26: 118–133.
- Lindenfeld J, Albert NM, Boehmer JP et al. Heart Failure Society of America. Comprehensive heart failure practice guideline. *J Card Fail*, 2010; 16: e1–e194.
- Freitas HF, Barbosa EA, Rosa FH et al. Association of HDL cholesterol and triglycerides with mortality in patients with heart failure. *J Med Biol Res*, 2009; 42: 420–425.
- Velagaleti RS, Massaro J, Vasan RS et al. Relations of lipid concentrations to heart failure incidence: the Framingham Heart Study. *Circulation*, 2009; 120: 2345–2351.
- Wang C, Harris WS, Chung M et al. n-3 Fatty acids from fish or fish-oil supplements, but not alpha-linolenic acid, benefit cardiovascular disease outcomes in primary-and secondary-prevention studies: a systematic review. *Am J Clin Nutr*, 2006; 84: 5–17.
- Masson S, Marchioli R, Mozaffarian D et al. Plasma n-3 polyunsaturated fatty acids in chronic heart failure in the GISSI-Heart Failure Trial: relation with fish intake, circulating biomarkers, and mortality. *Am Heart J*, 2013; 165: 208–15.e4.
- Doshi SN, McDowell IF, Moat SJ et al. Folic acid improves endothelial function in coronary artery disease via mechanisms largely independent of homocysteine lowering. *Circulation*, 2002; 105: 22–26.
- Usui M, Matsuoka H, Miyazaki H et al. Endothelial dysfunction by acute hyperhomocysteinemia: restoration by folic acid. *Clin Sci*, 1999; 96: 235–239.
- Durand P, Prost M, Blache D. Prothrombotic effects of folic acid deficient diet in rats platelets and macrophages related to elevated homocysteine and decreased n-3 polyunsaturated fatty acids. *Atherosclerosis*, 1996; 121: 231–243.
- Rautiainen S, Levitan EB, Mittleman MA et al. Total antioxidant capacity of diet and risk of heart failure: a population-based prospective cohort of women. *Am J Med*, 2013; 126: 494–500.

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15 marca 2014 roku

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PROGRAM

SESJA INAUGURACYJNA:

Jak poprawić wyniki leczenia niewydolności serca?
Stabilna choroba wieńcowa: wytyczne 2013
Przezkórne leczenie chorób zastawek
Bezdech/Czy rehabilitacja kardiologiczna leczy?

SESJA II: Lekarz rodzinny, internista, kardiolog — koalicja dla redukcji ryzyka

SESJA III: Nowe metody leczenia

SESJA IV: Co nowego w kardiologii: gorąca linia



Czy niewydolność serca determinuje sposób żywienia pacjentów?

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Streszczenie

Wstęp: Badania dotyczące sposobu żywienia pacjentów z niewydolnością serca (HF) dostępne w piśmiennictwie wskazują, że odbiega on w znacznym stopniu od zaleceń i norm żywienia.

Cel: Celem badania była ocena sposobu żywienia pacjentów z HF w średnim wieku w porównaniu z grupą kontrolną wyodrębnioną z populacji ogólnej bez HF.

Metody: Badaniem objęto 70 pacjentów z rozpoznaną HF, poniżej 65. rż., w I/II/III klasie wg NYHA. Do grupy kontrolnej włączono osoby z populacji ogólnej z identycznymi następującymi parametrami: płeć, wiek, wskaźnik masy ciała i kryterium palenia tytoniu jak badanych osób z HF. Sposób żywienia oceniono na podstawie kwestionariusza częstości spożycia oraz 24-godzinnego wywiadu żywieniowego.

Wyniki: Stosunek energia diety/idealna masa ciała był istotnie niższy w grupie badanej w porównaniu z grupą kontrolną ($p < 0,005$). W grupie badanej stwierdzono także istotnie niższy udział energii z tłuszczu, niższe spożycie nasyconych kwasów tłuszczowych, jednonienasyconych kwasów tłuszczowych ($p < 0,05$), kwasów omega-3 ($p < 0,005$), kwasu foliowego ($p < 0,001$) oraz błonnika pokarmowego ($p < 0,01$) w odniesieniu do grupy kontrolnej. Nie zaobserwowano różnic dotyczących spożycia witaminy B6, C, A i E między grupą badaną i kontrolną. Najniższe spożycie energii, błonnika pokarmowego i witamin (z wyjątkiem witaminy A) stwierdzono w grupie z HF w III klasie wg NYHA.

Wnioski: Niewydolność serca wiąże się ze zmianami w sposobie żywienia w porównaniu z osobami bez HF, które przejawiają się niższą energetycznością diety, niższą zawartością tłuszczu ogółem, szczególnie kwasów omega-3, a także niższym spożyciem kwasu foliowego i błonnika pokarmowego. Mimo że większość pacjentów charakteryzowała się etiologią niedokrwinną HF, a więc powinna stosować rekomendacje dietetyczne dla osób z dyslipidemią i wysokim ryzykiem sercowo-naczyniowym, ich dieta była niezgodna z zaleceniami (wysokie spożycie nasyconych kwasów tłuszczowych, niskie spożycie błonnika pokarmowego).

Słowa kluczowe: sposób żywienia, niewydolność serca, klasyfikacja wg NYHA, populacja ogólna

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