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Trabajo Original

Paciente crítico

The effects of a low-carbohydrate diet on oxygen saturation in heart failure patients: a randomized controlled clinical trial

Efecto de una dieta baja en hidratos de carbono sobre la saturación de oxígeno en pacientes con insuficiencia cardiaca: ensayo clínico aleatorizado

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Abstract

Introduction: Nutritional therapy in heart failure (HF) patients has been focused on fluid and sodium restriction with the aim of decreasing volume overload. However, these recommendations are not well established and sometimes controversial.

Objective: To evaluate the effect of the consumption of a low-carbohydrate diet on oxygen saturation, body composition and clinical variables during two months of follow-up in chronic, stable heart failure patients.

Methods: In a parallel group randomized controlled clinical trial, 88 ambulatory patients were randomly assigned to a low-carbohydrate diet group (40% carbohydrates, 20% protein and 40% fats [12% saturated, 18% monounsaturated and 10% polyunsaturated]) or a standard diet group (50% carbohydrates, 20% protein and 30% fats [10% saturated, 10% monounsaturated and 10% polyunsaturated]) for two months. Diets were normocaloric in both groups. At baseline and at two months of follow-up, the variables evaluated were: oxygen saturation, dietary intake, body composition and handgrip strength.

Results: After two months of follow-up, the low-carbohydrate diet group decreased the carbohydrate consumption and had improved oxygen saturation (93.0 ± 4.4 to 94.6 ± 3.2, p = 0.02), while the standard diet group had decreased (94.90 ± 2.4 to 94.0 ± 2.9, p = 0.03). There were also differences between the groups at the end of the study (p = 0.04). No significant differences showed in handgrip strength in both groups, low-carbohydrate diet group (26.4 ± 8.3 to 27.2 ± 8.3 kg, p = 0.07) and standard diet group (25.4 ± 8.9 to 26.1 ± 9.5 kg, p = 0.14).

Conclusions: Low-carbohydrate diet may improve the oxygen saturation in patients with chronic stable heart failure.

Key words:

Heart failure. Oximetry. Low carbohydrate diet. Unsaturated dietary fats. Clinical trial.

Resumen

Introducción: la terapia nutricional en pacientes con insuficiencia cardiaca (IC) ha sido enfocada en la restricción de líquidos y de sodio con el objetivo de reducir la sobrecarga de volumen. Sin embargo, estas recomendaciones no están bien establecidas y en algunos casos son controvertidas.

Objetivo: evaluar el efecto del consumo de una dieta baja en hidratos de carbono sobre la saturación de oxígeno, composición corporal y variables clínicas durante dos meses de seguimiento en pacientes con insuficiencia cardiaca estable.

Métodos: ensayo clínico aleatorizado paralelo en 88 pacientes ambulatorios que fueron asignados aleatoriamente al grupo dieta baja en hidratos de carbono (40% hidratos de carbono, 20% proteínas y 40% lípidos [12% saturadas, 18% monoinsaturadas y 10% poliinsaturadas]) o al grupo dieta estándar (50% hidratos de carbono, 20% proteínas y 30% lípidos [10% saturadas, 10% monoinsaturadas y 10% poliinsaturadas]) por dos meses. Las dietas fueron normocalóricas en ambos grupos. En la medición basal y a los dos meses de seguimiento, las variables evaluadas fueron: saturación de oxígeno, ingesta dietética, composición corporal y fuerza de prensión de mano.

Resultados: después de dos meses de seguimiento, el grupo de dieta baja en hidratos de carbono disminuyó el consumo de hidratos de carbono y mejoró la saturación de oxígeno (93.0 ± 4.4 to 94.6 ± 3.2, p = 0.02), mientras que el grupo de dieta estándar disminuyó (94.90 ± 2.4 to 94.0 ± 2.9, p = 0.03). También se observó diferencia entre los grupos al final del estudio (p = 0.04). No se observaron diferencias estadísticamente significativas en fuerza de mano en ambos grupos: dieta baja en hidratos de carbono (26.4 ± 8.3 to 27.2 ± 8.3 kg, p = 0.07) y dieta estándar (25.4 ± 8.9 to 26.1 ± 9.5 kg, p = 0.14).

Conclusiones: la dieta baja en hidratos de carbono mejora la saturación de oxígeno en pacientes con insuficiencia cardiaca estable.

Palabras clave:

Insuficiencia cardiaca. Oximetría. Dieta baja en hidratos de carbono. Lípidos dietéticos insaturados. Ensayo clínico.

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INTRODUCTION

Heart failure (HF) is a complex syndrome with high morbidity and mortality. One of the main causes of decompensation is non-compliance with diet. In HF patients, nutritional therapy has been focused on fluid and sodium restriction with the aim of decreasing volume overload (1). However, those recommendations are not well established and sometimes controversial (2-6).

Another purpose of nutritional therapy is to reduce cardiovascular risk, and some studies suggest that saturated fatty acids should be replaced by some other macronutrient, such as monounsaturated fatty acids. This change has been associated with decreased total cholesterol, LDL cholesterol, and HDL cholesterol, while the replacement of saturated fatty acids with carbohydrates showed decreased total cholesterol, LDL cholesterol, and HDL cholesterol but increased triacylglycerols (7).

Moderately low-carbohydrate and high-unsaturated-fat diets have been demonstrated to improve blood pressure (8), decrease triacylglycerols, total cholesterol (8,9) and heart rate (8), enhance insulin sensitivity (8,10), improve endothelial function (9), reduce the waist circumference (9) and, finally, decrease cardiovascular risk and total mortality (11,12) in other populations.

In the case of HF patients, reducing cardiovascular risk through the replacement of carbohydrates and saturated fatty acids with monounsaturated fatty acids may also improve clinical status. The Mediterranean diet, which is moderately low in carbohydrates and monounsaturated and alpha-linolenic acid-rich diet, was associated with improved systolic function and left ventricular filling pressure in HF patients (13) as well as diminished cardiovascular risk, HF biomarkers, terminal pro-brain natriuretic peptide, oxidized LDL and lipoprotein (a) (14).

Carbohydrates have the highest respiratory quotient (ratio of carbon dioxide production to oxygen consumption) of the macronutrients and consequently increase the carbon dioxide (VCO_2) to be metabolized. In subjects with decreased respiratory function, this results in signs of increased breathing work. In other populations, a low-carbohydrate and high-fat diet has been associated with greater respiratory efficiency (15-17), improved arterial carbon dioxide tension ($PaCO_2$), arterial oxygen tension (PaO_2) and oxygen saturation (SaO_2), decreased respiratory quotient (RQ) as well as improved lung function, with increased forced expiratory volume (FEV_1) and oxygen consumption (VO_2) and reduced minute ventilation (VE) and VCO_2 (15,17,18).

On the other hand, HF is considered to be a complex condition in which the heart cannot pump adequate oxygen-rich blood to satisfy the body requirements, peak oxygen consumption is diminished with lower respiratory efficiency and, finally, lung function and ventilator inefficiency (19,20).

In spite of the above mentioned observations, nutritional therapy of HF patients has not been focused on improving mechanical ventilation by optimizing the consumption of oxygen to be metabolized by diet. Therefore, the aim of the present study was to assess the effect of a low-carbohydrate fat diet on oxygen saturation, body composition and clinical variables of patients with chronic stable HF, and to assess the association between dietary intake and changes in SaO_2 at the end of the study. Our hypothesis was that HF patients assigned to the low-carbohydrate diet group would have improved SaO_2 in comparison with the standard diet group.

METHODS AND MATERIALS

STUDY DESIGN

This was a parallel group randomized controlled clinical trial in 88 ambulatory patients of the Heart Failure Clinic at the Instituto Nacional de Ciencias Médicas y Nutrición "Salvador Zubiran" (INCMNSZ). The study was approved by the Institutional Ethics Committee of Biomedical Research in Humans of the INCMNSZ, and in accordance with the World Medical Association and the Helsinki Declaration. The subjects were informed about the aims of the study, and the patients who agreed to participate signed an informed consent form.

PARTICIPANTS

Eligibility criteria were: patients with a confirmed diagnosis of HF and classified according to European Society of Cardiology (ESC) (1), in stable New York Heart Association (NYHA) functional class I to III, 18 years of age or older. Exclusion criteria were: NYHA functional class IV, patients in severe renal failure (glomerular filtration rate < 30 ml/min/1.73 m^2), liver failure, and cancer. The first patient was enrolled on May 5, 2011, and the last patient completed the intervention on December 13, 2013.

RANDOMIZATION AND BLINDING

After baseline measurements, subjects were randomly assigned to the low-carbohydrate group (n = 45) or the standard diet group (n = 43) using a randomized sequence created on the website http://www.randomization.com. The patients and the nutritionist who assigned treatment with a sequentially numbered list were aware of group assignment, however the cardiologist or study collaborators who performed the evaluations were blinded to the assigned diet.

INTERVENTIONS

The macronutrient compositions of the low-carbohydrate group was 40% carbohydrates, 40% fats (12% saturated, 18% monounsaturated and 10% polyunsaturated) and 20% protein; the standard diet group composition was designed according to the recommendations of the American Heart Association Dietary Guidelines (21): 50% carbohydrates, 30% fats (10% saturated, 10% monounsaturated and 10% polyunsaturated) and 20% protein. Diets were normocaloric in both groups. The normocaloric

diet was calculated according for each patients in both groups. The basal energy expenditure of subjects was estimating using Harris and Benedict´s equation.

All patients received a nutritional handbook and oral instructions individually from the dietitian about the nutritional treatment to which they were assigned, and sodium and fluid restriction followed established guidelines (1,22). The nutritional handbook contained seven menus from which patients could choose to cook and consume during the study. Patients and their family members were scheduled two visits at one and two months at which the study collaborators could answer questions about their nutritional treatment and assess the degree of compliance with the nutritional treatment using a 24 h food recall. The patients in both groups received recommended guidelines pharmacological HF treatment by cardiologists.

OUTCOMES

The primary objective was to assess the effect of low-carbohydrate and standard diet on oxygen saturation. The second objective evaluated was the impact on body composition and clinical variables: weight, third space water, impedance index, phase angle, total body and extracellular water, handgrip strength and systolic and diastolic blood pressures. The third objective was to assess the association between dietary intake and changes in SaO_2 at the end of the study. The variables were evaluated at baseline and two months of follow-up.

MEASUREMENTS

Oxygen saturation (SaO_2) was measured using the analog pulse finger oximeter (MD200, Hergom). The SaO_2 was taken on seated subjects on the index finger without enamel after one minute.

Change in SaO_2 was obtained as follows: change in SaO_2 = ([end SaO_2 (%) - baseline SaO_2/baseline SaO_2]*100). Dietary intake and compliance with diet were assessed by 24 h food recall using ESHA Food Processor SQL software (version 7.9, ESHA Research, Salem. OR 2001). With regard to body composition, weight and height were measured according to the manual reference of anthropometric standardization (23); all subjects wore light clothing and were barefoot. The variables third space water, impedance index, phase angle, total body and extracellular water were evaluated by bioelectrical impedance analysis (BIA), using a tetra-polar and multiple-frequency equipment (BodyStat Quad-Scan 4000, Bodystat Ltd.; Isle of Man, UK). Before undergoing bioimpedance, the subjects were placed in supine position with their legs and arms in 30° abduction position for ≈ 10 minutes. Handgrip strength was measured using the Hand Grip Dynamometer Analogue (Takei 5001). Patients were instructed to apply as much handgrip pressure as possible with their right and left hands. The measurements were repeated twice with each hand, and the highest score was recorded in kilograms (24). Systolic and diastolic pressure was evaluated by the Automatic Blood Pressure Monitor (Microlife BP 3BT0-AP).

SAMPLE SIZE

Sample size was calculated using a formula for comparison of means in two independent samples with significance level of 0.05 and power of 0.80. We used the change in the SaO_2 between the study groups reported by Borghi-Silva et al. as the reference (25). The change in SaO_2 over six weeks was 5% in the L-carnitine group and 3% in the standard group, and the standard deviation was 7. A sample size of 34, which was increased by 20% lost to follow up, gave a total of 41 patients in each group.

STATISTICAL METHODS

All data were analyzed using STATA/SE version 12.0 (STATA Corporation, College Station, TX). Results of continuous variables were expressed as mean ± standard deviation (SD); those with an asymmetrical distribution, as median (25th and 75th); and categorical variables, as absolute and relative frequency. The comparisons of variables at baseline and at the end of the study between the groups were analyzed with χ^2 (categorical variables) and paired Student's t-test or Wilcoxon (continuous variables). The comparisons between the groups at the end of the study were evaluated as the percentage change ([end variable - baseline variable / baseline variable]*100) between baseline and the end of study after they were analyzed with unpaired Student's t-test or U de Mann Whitney (continuous variables). Subsequently, the association between changes in carbohydrate intake and changes in SaO_2 was obtained with Pearson correlation. A p value < 0.05 was considered as statistically significant.

RESULTS

A total of 88 ambulatory patients met the inclusion criteria and were included in the study. The subjects were randomized to the low-carbohydrate diet group (n = 45) or standard diet group (n = 43). Of these patients, 38 and 35, respectively, completed the follow-up (Fig. 1).

With respect to baseline characteristics of participants in both groups who completed and did not complete the nutritional intervention, within the low carbohydrate diet group, the subjects who did not complete were younger than the subjects who completed the intervention (57 ± 12.98 *versus* 68 ± 12.65, p = 0.03, respectively). In relation to other variables, non-significant differences were observed in both study groups between the subjects who complete and those who did not complete the intervention (data not shown).

As far as subjects who completed the intervention were concerned, no significant differences were found in the baseline characteristics between the study groups. In both groups there was a high prevalence of diabetes, dyslipidemia, obesity, hypertension and cachexia. The highest proportion of subjects was found in NYHA functional class I (Table I). In addition, all patients of the low-carbohydrate diet group and standard diet group

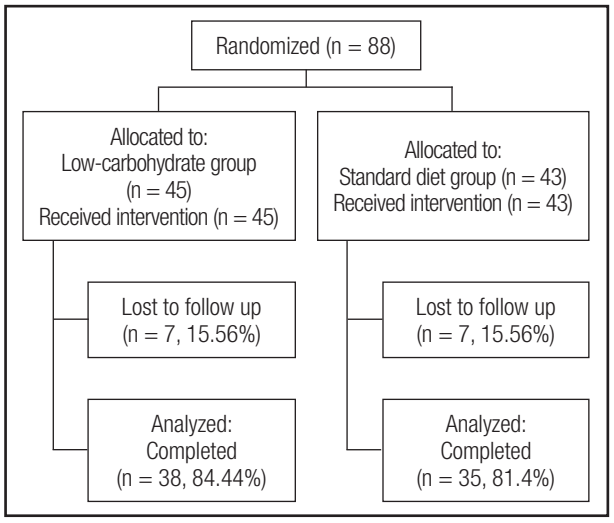

Figure 1.

Process flow diagram of subjects who were included in trial.

were on conventional pharmacological management of HF, and there were no statistically significant differences between the groups. No changes in pharmacological treatment after two months were observed. After two months of follow-up, patients in the low-carbohydrate diet group reported a decrease in the consumption of carbohydrates (51.4 ± 6.1 to 42.3 ± 2.8, p = 0.05), and the standard diet group showed an increase (52.3 ± 15.2 to 54.3 ± 2.9, p = 0.74). Differences between the study groups were found at the end of the study (p = 0.04). With respect to sodium intake, both study groups showed a decreased intake, with no statistically significant differences between the groups after two months of follow-up. Fiber intake also increased in the standard diet group with no statistically significant difference (Table II).

Table III shows the comparison of clinical and body composition variables. We can see that the SaO_2 changed after two months of follow-up; in the low-carbohydrate group it increased from 93.0 ± 4.4 to 94.6 ± 3.18, p = 0.02, while in the standard diet group it decreased from 94.9 ± 2.4 to 94 ± 2.92, p = 0.03. Differences between the groups were also found at the end of the study (p = 0.004). No statistically significant differences with respect to systolic and diastolic blood pressure between the two groups were observed after two months of follow-up. In the category of body composition variables, non-significant differences were observed in weight, third space water, phase angle, total body water or extracellular water after two months of follow-up within the groups. With respect to handgrip strength, no significant differences were showed in both groups, low-carbohydrate diet group (26.4 ± 8.3 to 27.2 ± 8.3 kg, p = 0.07) and standard group (25.4 ± 8.9 to 26.1 ± 9.5 kg, p = 0.14) after two months of follow-up. No statistically significant differences were observed in clinical and body composition between the groups at the end of the study. Also, inverse correlation between changes in carbohydrate intake and changes in SaO_2 was found (r = -0.41, p = 0.014).

Table I. Baseline characteristics of participants who completed the intervention

Variables	Standard diet group n = 43	Low-carbohydrate group n = 45	p-value
Male (n [%])	16 (45.71)	19 (50)	0.71
Age (y)	70.45 ± 12.35	68.47 ± 12.65	0.50
Systolic HF (n [%])	15 (45.45)	16 (42.11)	0.88
Diastolic HF (n [%])	6 (18.18)	10 (26.32)	
Systolic and diastolic HF (n [%])	9 (27.27)	9 (23.68)	
Diabetes (n [%])	19 (54.29)	20 (52.63)	0.89
Obesity (n [%])	10 (28.57)	17 (44.74)	0.15
Dyslipidemia (n [%])	12 (34.29)	9 (23.68)	0.32
Hypertension (n [%])	27 (77.14)	29 (76.32)	0.93
Renal failure (n [%])	6 (17.14)	6 (15.79)	0.88
Cachexia (n [%])	19 (54.29)	15 (39.47)	0.21
NYHA functional class			
NYHA I (n [%])	22 (62.86)	25 (71.43)	0.72
NYHA II (n [%])	11 (31.43)	8 (22.86)	
NYHA III (n [%])	2 (5.71)	2 (5.71)	

HF: Heart failure; NYHA: New York Heart Association. The data are presented as percentage or mean ± standard deviation.

Table II. Comparison of nutrient intake from baseline to end of the study

	Diet group	Baseline	End	p value within groups	% change	p value between groups
Energy intake (kcal)	Low-carbohydrate	1,840.2 ± 510.6	1,327.1 ± 235.0	0.10	-23.3 ± 23.8	0.11
	Standard	1,605.9 ± 478.6	1,460.4 ± 235.0	0.13	-6.5 ± 11.7	
Carbohydrates (%)	Low-carbohydrate	51.4 ± 6.1	42.3 ± 2.8	0.05	-15.1 ± 9.8	0.04
	Standard	52.3 ± 15.2	54.3 ± 2.9	0.74	14.1 ± 43.4	
Protein (%)	Low-carbohydrate	19 ± 8.2	19.5 ± 3.5	0.98	13.5 ± 41.4	0.27
	Standard	20.2 ± 5.9	17.2 ± 4.5	0.20	-9.7 ± 31.1	
Fats (%)	Low-carbohydrate	26.5 ± 13.5	24.3 ± 10.2	0.47	17.7 ± 74.0	0.90
	Standard	25.7 ± 14.9	26.5 ± 16.8	0.76	15.6 ± 60.6	
Fiber (g)	Low-carbohydrate	22.3 ± 7.2	18.0 ± 6.9	0.19	-14.8 ± 20.7	0.14
	Standard	16.8 ± 9.1	21.9 ± 6.1	0.21	101.4 ± 211.3	
Sodium (mg)	Low-carbohydrate	2,592.8 ± 1,484.4	1,505.6 ± 659.3	0.28	-0.4 ± 99.8	0.89
	Standard	2,121.1 ± 973.5	1,998.2 ± 919.8	0.66	4.87 ± 48.8	

The data are presented as percentage or mean ± standard deviation.

Table III. Comparison of body composition and clinical variables from baseline to end of the study

Variables	n	Baseline	2 months	p-value within group	% change	p for % change between groups
Oxygen saturation (%)						
Low-carbohydrate diet group	35	93.0 ± 4.4	94.6 ± 3.2	0.02	1.86 ± 4.82	0.004
Standard diet group	31	94.9 ± 2.4	94.0 ± 2.9	0.03	-0.97 ± 2.46	
SBP (mmHg)						
Low-carbohydrate diet group	32	123.5 ± 20.4	118.6 ± 19.8	0.20	-2.67 ± 17.11	0.385
Standard diet group	33	126.5 ± 21.3	125.5 ± 16.6	0.78	0.83 ± 2.65	
DBP (mmHg)						
Low-carbohydrate diet group	32	71.1 ± 10.9	70.7 ± 13.4	0.88	-1.42 ± 11.37	0.519
Standard diet group	33	72.3 ± 12.8	70.3 ± 8.7	0.18	2.02 ± 27.87	
Weight (kg)						
Low-carbohydrate diet group	29	92.4 ± 30.5	86.1 ± 27. 0	0.34	-0.78 ± 3.70	0.145
Standard diet group	33	85.7 ± 29.9	88.3 ± 28.9	0.58	0.27 ± 2.09	
Phase angle						
Low-carbohydrate diet group	37	5.8 ± 1.2	5.8 ± 1.2	0.27	3.30 ± 33.28	0.546
Standard diet group	32	5.2 ± 1.1	5.4 ± 0.9	0.21	7.69 ± 25.74	
TBW (%)						
Low-carbohydrate diet group	35	51.5 ± 7.7	49.22 ± 9.0	0.27	1.10 ± 4.99	0.322
Standard diet group	29	52.0 ± 7.6	50.68 ± 7.6	0.21	5.52 ± 25.62	
ECW (%)						
Low-carbohydrate diet group	28	23.0 ± 2.8	22.89 ± 2.7	0.54	0.55 ± 3.81	0.502
Standard diet group	34	23.1 ± 2.6	22.81 ± 2.6	0.72	-0.20 ± 5.10	
Handgrip strength (kg)						
Low-carbohydrate diet group	31	26.4 ± 8.3	27.2 ± 8.3	0.07	4.16 ± 10.71	0.712
Standard diet group	35	25.4 ± 8.9	26.1 ± 9.5	0.14	3.17 ± 11.03	

SBP: Systolic blood pressure; DBP: Diastolic blood pressure; TBW: Total body water; ECW: Extracellular water. The data with symmetrical distribution are presented as mean ± standard deviation.

DISCUSSION

This controlled clinical study showed the effects of a low-carbohydrate diet on the SaO_2 in chronic stable heart failure patients. The principal finding was enhanced in SaO_2 in the low-carbohydrate group, while in the standard diet group the SaO_2 decreased after two months of follow-up. Thus, oxygen saturation at the end of treatment showed statistically significant differences between the study groups. As far as the effect of dietary intake on SaO_2 in chronic obstructive pulmonary disease (COPD) is concerned, SaO_2 has been observed to decrease immediately after food intake (16), and this decline becomes more accentuated with greater severity of pulmonary dysfunction (26). Moreover, in a clinical trial, Borghi-Silva et al. showed that supplementation with two grams of L-carnitine daily and exercise was associated with a statistically significant increase in SaO_2, while the placebo-exercise group did not have a significant increase after six weeks of follow-up (25). Another clinical trial compared the effect of different macronutrients on SaO_2 with two supplements: low-carbohydrate/high-fat supplements *versus* high-carbohydrate/low-fat. The study showed that SaO_2 decreased at 10 and 15 minutes after high-carbohydrate/low-fat supplement intake, while no changes occurred with low-carbohydrate/high-fat supplements (16). Similarly, the proportion of carbohydrate, lipid and protein intake has an effect on respiratory gas exchange. Several studies have shown that high carbohydrate diets are correlated with increased VO_2, VCO_2 and CR both in healthy subjects and in COPD patients (15,18), as well as increased VE and Borg scale with diminished exercise tolerance (15,16) compared with low-carbohydrate and high fat diets. The adverse effects on respiratory efficiency after high-carbohydrate intake are due to the fact that absorption and metabolism of carbohydrate intake has more VCO_2 produced per molecule of VO_2 and QR than those of fatty acids or protein, increasing the ventilatory response. This suggests that macronutrient distribution changes in diet can improve respiratory gas exchange (15,16,18), respiratory function (16) and exercise tolerance (15,16,27).

The effect of this macronutrient distribution has not been studied in patients with HF with the aim of optimizing the ventilatory response. HF patients are characterized by impaired cardiopulmonary and pulmonary function with reduced peak oxygen consumption and oxygen saturation (28,32). Decreased peak oxygen consumption correlates with oxygen saturation and lower muscle mass. It is also associated with less muscle strength and diminished exercise tolerance (28,29,31-33). The peak oxygen consumption, muscle strength, VE/VCO_2 and oxygen saturation are independent predictors of hospitalization and mortality in HF patients (18,33-35).

The subjects had a positive adherence. In the intervention group the low-carbohydrate diet composition was 40% carbohydrates of total intake and the intervention group had a decrease in carbohydrate intake from 51 to 42%. However, in the standard group the diet composition was 50% carbohydrate. The carbohydrate intake in the control group was 54% at the end of the study. Moreover, the study groups were analyzed with the intention-to-treat principle, in which all subjects were evaluated according to their random group assignment, regardless of the degree of patient compliance. This approach does not permit overestimation of the effects of therapy in the study and the intervention is probably more effective in patients who adhere to nutritional therapy (36).

Others researchers have reported associations between food groups and types of nutrient intake on cardiopulmonary and pulmonary function variables. In a cohort study, Root et al. observed a positive association of FEV(1)/FVC with intake of whole grains, animal protein, dietary fiber, polyunsaturated fatty acids and omega-3 fatty acids, while saturated fatty acids as well as solid fats, alcohol, and added sugar intake showed a negative association with lung function (37). Furthermore, solid fats, alcohol, and added sugar intake were analyzed together, and this did not make it possible to evaluate the effect of sugar intake on pulmonary function. In our study, no statistically significant differences were observed in the effects of the rest of the macronutrients, possibly due to the limited sample size.

Although the literature shows the effect of carbohydrates on gas exchange, neither the effect of the carbohydrate type on oxygen saturation nor the effect of different macronutrient distributions on oxygen saturation in patients with HF have been studied. On the other hand, in our study, the low-carbohydrate diet group did not show increased handgrip strength possibly due to limited follow-up time. However, more experimental studies with longer follow-ups that assess the effectiveness of the low-carbohydrate nutritional intervention on muscular strength are necessary. Other nutritional therapy has also been shown to maintain or improve handgrip strength (38,39), which is considered as an indicator of muscular function with prognostic implications (33).

This evidence should help to develop a nutritional treatment according to the needs of HF patients, although more clinical trials to assess the effect of different proportions of macronutrients on pulmonary and cardiorespiratory function are warranted. The quality and quantity of the types of nutrients consumed are also important. The majority of the nutritional recommendations for heart failure patients come from observational studies or guidelines for reducing the risk of cardiovascular disease, but these guidelines are designed for the general population.

STUDY LIMITATIONS

The study has some limitations. First, the intervention period was short. A longer follow-up would probably show changes in such variables as body composition and systolic and diastolic pressure. Second, as patients were ambulatory we had high rates of patient's lost-to-follow-up.

STUDY STRENGTHS

Low-carbohydrate nutritional therapy can be easily reproduced in real life and can be prepared at home without difficulty.

CONCLUSIONS

A low-carbohydrate diet may increase the SaO_2 in chronic stable heart failure patients.

REFERENCES

1. McMurray JJV, Adamopoulos S, Anker SD, Auricchio A, Böhm M, Dickstein K, et al. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart. Rev Port Cardiol 2013;32:e1-641-e61. DOI: 10.1093/eurheartj/ehs104.
2. Kollipara UK, Jaffer O, Amin A, Toto KH, Nelson LL, Schneider R, et al. Relation of lack of knowledge about dietary sodium to hospital readmission in patients with heart failure. Am J Cardiol 2008;102(9):1212-5. E-pub 2008/10/23. DOI: 10.1016/j.amjcard.2008.06.047.
3. Butler J, Casey DE, Drazner MH, Fonarow GC, Geraci Sa, Horwich T, et al. 2013 ACCF/AHA Guideline for the Management of Heart Failure: Executive Summary. JAC 2013;62:1495-539. DOI: 10.1016/j.jacc.2013.05.020.
4. Hummel SL, DeFranco AC, Skorcz S, Montoye CK, Koelling TM. Recommendation of low-salt diet and short-term outcomes in heart failure with preserved systolic function. Am J Med 2009;122(11):1029-36. DOI: 10.1016/j.amjmed.2009.04.025.
5. Lennie TA, Song EK, Wu JR, Chung ML, Dunbar SB, Pressler SJ, et al. Three gram sodium intake is associated with longer event-free survival only in patients with advanced heart failure. J Card Fail 2011;17:325-30. DOI: S1071-9164(10)01228-5 [pii]\n10.1016/j.cardfail.2010.11.008.
6. Colin-Ramírez E, McAlister FA, Zheng Y, Sharma S, Armstrong PW, Ezekowitz JA. The long-term effects of dietary sodium restriction on clinical outcomes in patients with heart failure. The SODIUM-HF (Study of Dietary Intervention under 100 mmol in Heart Failure): A pilot study. Am Heart J 2015;169(2):274-81e1. DOI: 10.1016/j.ahj.2014.11.013.
7. Berglund L, Lefevre M, Ginsberg HN, Kris-Etherton PM, Elmer PJ, Stewart PW, et al. Comparison of monounsaturated fat with carbohydrates as a replacement for saturated fat in subjects with a high metabolic risk profile: Studies in the fasting and postprandial states. Am J Clin Nutr 2007;86(6):1611-20.
8. Muzio F, Mondazzi L, Harris WS, Sommariva D, Branchi A. Effects of moderate variations in the macronutrient content of the diet on cardiovascular disease risk factors in obese patients with the metabolic syndrome. Am J Clin Nutr 2007;86(4):946-51.
9. Somerseta SM, Graham L, Markwell K. Isoenergetic replacement of dietary saturated with monounsaturated fat via macadamia nuts enhances endothelial function in overweight subjects. e-SPEN J 2013;8:e113- e9. DOI: 10.1016/j.clnme.2013.02.003.
10. Maggioni AP, Dahlstrom U, Filippatos G, Chioncel O, Leiro MC, Drozdz J, et al. EURObservational Research Programme: the Heart Failure Pilot Survey (ESC-HF Pilot). Eur J Heart Fail 2010;12(10):1076-84. DOI: 10.1093/eurjhf/hfq154.
11. Halton TL, Willett WC, Liu S, Manson JE, Albert CM, Rexrode K, et al. Low-carbohydrate-diet score and the risk of coronary heart disease in women. N Engl J Med 2006;355(19):1991-2002. DOI: 10.1056/NEJMoa055317.
12. Nakamura Y, Okuda N, Okamura T, Kadota A, Miyagawa N, Hayakawa T, et al. Low-carbohydrate diets and cardiovascular and total mortality in Japanese: A 29-year follow-up of NIPPON DATA80. Br J Nutr 2014;112(6):916-24. DOI: 10.1017/S0007114514001627.
13. Chrysohoou C, Pitsavos C, Metallinos G, Antoniou C, Oikonomou E, Kotroyiannis I, et al. Cross-sectional relationship of a Mediterranean type diet to diastolic heart function in chronic heart failure patients. Heart Vessels 2012;27:576-84. DOI: 10.1007/s00380-011-0190-9.
14. Fito M, Estruch R, Salas-Salvado J, Martínez-González MA, Aros F, Vila J, et al. Effect of the Mediterranean diet on heart failure biomarkers: A randomized sample from the PREDIMED trial. Eur J Heart Fail 2014;16(5):543-50. DOI: 10.1002/ejhf.61.
15. Efthimiou J, Mounsey PJ, Benson DN, Madgwick R, Coles SJ, Benson MK. Effect of carbohydrate rich versus fat rich loads on gas exchange and walking performance in patients with chronic obstructive lung disease. Thorax 1992;47(6):451-6.
16. Vermeeren MA, Wouters EF, Nelissen LH, Van Lier A, Hofman Z, Schols AM. Acute effects of different nutritional supplements on symptoms and functional capacity in patients with chronic obstructive pulmonary disease. Am J Clin Nutr 2001;73(2):295-301.
17. Cai B, Zhu Y, Ma Yi, Xu Z, Zao Yi, Wang J, et al. Effect of supplementing a high-fat, low-carbohydrate enteral formula in COPD patients. Nutrition 2003;19(3):229-32. DOI: 10.1016/s0899-9007(02)01064-x.
18. Kuo CD, Shiao GM, Lee JD. The effects of high-fat and high-carbohydrate diet loads on gas exchange and ventilation in COPD patients and normal subjects. Chest 1993;104(1):189-96.
19. Ponikowski P, Francis DP, Piepoli MF, Davies LC, Chua TP, Davos CH, et al. Enhanced ventilatory response to exercise in patients with chronic heart failure and preserved exercise tolerance: Marker of abnormal cardiorespiratory reflex control and predictor of poor prognosis. Circulation 2001;103(7):967-72.
20. Gosker HR, Lencer NHMK, Franssen FME, Van Der Vusse GJ, Wouters EFM, Schols AMWJ. Striking similarities in systemic factors contributing to decreased exercise capacity in patients with severe chronic heart failure or COPD. Chest 2003;123:1416-24. DOI: 10.1378/chest.123.5.1416
21. Krauss RM, Eckel RH, Howard B, Appel LJ, Daniels SR, Deckelbaum RJ, et al. AHA Dietary Guidelines. Revision 2000: A statement for healthcare professionals from the Nutrition Committee of the American Heart Association. Circulation 2000;102(18):2284-99.
22. Yancy CW, Jessup M, Bozkurt B, Masoudi FA, Butler J, McBride PE, et al. 2013 ACCF/AHA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am College Cardiol 2013. DOI: 10.1016/j.jacc.2013.05.019.
23. Lohman TG RA, Martorell R. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics; 1991.
24. Rantanen T, Guralnik JM, Foley D, Masaki K, Leveille S, Curb JD, et al. Midlife hand grip strength as a predictor of old age disability. JAMA 1999; 281(6):558-60.
25. Borghi-Silva A, Baldissera V, Sampaio LM, Pires-DiLorenzo VA, Jamami M, Demonte A, et al. L-carnitine as an ergogenic aid for patients with chronic obstructive pulmonary disease submitted to whole-body and respiratory muscle training programs. Braz J Med Biol Res 2006;39(4):465-74. DOI: / S0100-879x2006000400006.
26. Wolkove N, Fu LY, Purohit A, Colacone A, Kreisman H. Meal-induced oxygen desaturation and dyspnea in chronic obstructive pulmonary disease. Can Respir J 1998;5(5):361-5.
27. Frankfort JD. Effects of high- and low-carbohydrate meals on maximum exercise performance in chronic airflow obstruction. CHEST J 1991;100(3):792. DOI: 10.1378/chest.100.3.792.
28. Chua TP, Ponikowski P, Harrington D, Anker SD, Webb-Peploe K, Clark AL, et al. Clinical correlates and prognostic significance of the ventilatory response to exercise in chronic heart failure. J Am Coll Cardiol 1997;29(7):1585-90.
29. Cicoira M, Zanolla L, Franceschini L, Rossi A, Golia G, Zamboni M, et al. Skeletal muscle mass independently predicts peak oxygen consumption and ventilatory response during exercise in noncachectic patients with chronic heart failure. J Am Coll Cardiol 2001;37(8):2080-5.
30. Agostoni PG, Bussotti M, Palermo P, Guazzi M. Does lung diffusion impairment affect exercise capacity in patients with heart failure? Heart 2002;88(5):453-9.
31. Gosker HR. Striking similarities in systemic factors contributing to decreased exercise capacity in patients with severe chronic heart failure or COPD. Chest 2003;123(5):1416-24. DOI: 10.1378/chest.123.5.1416.
32. Manetos C, Dimopoulos S, Tzanis G, Vakrou S, Tasoulis A, Kapelios C, et al. Skeletal muscle microcirculatory abnormalities are associated with exercise intolerance, ventilatory inefficiency, and impaired autonomic control in heart failure. J Heart Lung Transplant 2011;30(12):1403-8. DOI: 10.1016/j.healun.2011.08.020.
33. Izawa KP, Watanabe S, Osada N, Kasahara Y, Yokoyama H, Hiraki K, et al. Handgrip strength as a predictor of prognosis in Japanese patients with congestive heart failure. Eur J Cardiovasc Prev Rehabil 2009;16(1):21-7. DOI: 10.1097/HJR.0b013e32831269a3.
34. Nakanishi M, Takaki H, Kumasaka R, Arakawa T, Noguchi T, Sugimachi M, et al. Targeting of high peak respiratory exchange ratio is safe and enhances the prognostic power of peak oxygen uptake for heart failure patients. Circul J 2014;78(9):2268-75.
35. Ponikowski P, Francis DP, Piepoli MF, Davies LC, Chua TP, Davos CH, et al. Enhanced ventilatory response to exercise in patients with chronic heart failure and preserved exercise tolerance: Marker of abnormal cardiorespiratory reflex control and predictor of poor prognosis. Circulation 2001;103(7):967-72.
36. Capurro D, Gabrielli L, Letelier LM. Intention to treat and follow up are important in assessing validity of a randomized clinical trial. Rev Méd Chile 2004;132(12):1557-60.
37. Root MM, Houser SM, Anderson JJ, Dawson HR. Healthy Eating Index 2005 and selected macronutrients are correlated with improved lung function in humans. Nutr Res 2014;34(4):277-84. DOI: 10.1016/j.nutres.2014.02.008.
38. Robinson SM, Jameson KA, Batelaan SF, Martin HJ, Syddall HE, Dennison EM, et al. Diet and its relationship with grip strength in community-dwelling older men and women: The Hertfordshire cohort study. J Am Geriatr Soc 2008;56(1):84-90. DOI: 10.1111/j.1532-5415.2007.01478.x.
39. Rahi B, Morais JA, Dionne IJ, Gaudreau P, Payette H, Shatenstein B. The combined effects of diet quality and physical activity on maintenance of muscle strength among diabetic older adults from the NuAge cohort. Exp Gerontol 2014;49:40-6. DOI: 10.1016/j.exger.2013.11.002.