Nutrition 30 (2014) 1366–1371

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Applied nutritional investigation

Dietary fatty acids intake and mortality in patients with heart failure

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ARTICLE INFO

Article history:
Received 11 January 2014
Accepted 2 April 2014

Keywords:
Dietary intake
dietary fats
nutrition
diet

ABSTRACT

Objective: Dietary strategies in heart failure (HF) are focused on sodium and fluid restriction to minimize the risk for acute volume overload episodes. However, the importance of dietary factors beyond sodium intake in the prognosis of the disease is uncertain. The purpose of this study was to evaluate the association of macro- and micronutrients intake on 1-y mortality in patients with HF.
Methods: A secondary analysis of 203 patients with chronic HF enrolled in a randomized trial of sodium reduction was completed. Patients with a complete 3-d food record at baseline were included in this analysis (N = 118); both control and intervention arms were combined. Three-d mean dietary intake was estimated. Cox multivariable regression analysis was used to evaluate the association between dietary factors and 1-y mortality.
Results: Among the 118 included patients, 54% were men, median (25th–75th percentiles) age 66 y (52–75 y), median ejection fraction 45% (30%–60%), and ischemic etiology present in 49% of patients. The association with 1-y mortality was significant for both polyunsaturated fatty acids (PUFA; adjusted hazard ratio [HR], 0.67; 95% confidence interval [CI]. 0.51–0.86 for intake as percentage of daily energy) and saturated fatty acids (SFA; adjusted HR, 1.15; 95% CI, 1.03–1.30 for intake as percentage of daily energy). Median of intake as percentage of daily energy was 5.3% for PUFAs and 8.2% for SFAs.
Conclusions: Intake of PUFAs and SFAs was independently associated with 1-y all-cause mortality in patients with chronic HF. Limiting dietary SFA and increasing PUFA intake may be advisable in this population.

Introduction

Heart failure (HF) is a common and growing public health problem worldwide. In the United States, approximately 5 million individuals have HF, and >550 000 patients are diagnosed with HF for the first time each year [1]. Despite advances in pharmacologic and nonpharmacologic therapies in patients with HF, mortality and morbidity remains significantly high [2,3].

Poor nutritional status has been reported to have a strong association with worse survival in these patients [4], and a compelling body of evidence suggests a potential role of micronutrients deficiencies in HF [5,6]. However, the association between macronutrient intake, including dietary fats, and prognosis in the HF population has not been well studied. A recent

ECR is supported by a postdoctoral fellowship from Alberta Innovates–Health Solutions and the National Council of Science and Technology (CONACYT), Mexico. JAE holds a population health investigator award from Alberta Innovates–Health Solutions. ECR was responsible for the conception and design of the study; generation, collection, and interpretation of data; and drafting the manuscript. LCM and AOT were responsible for generation of data and revision of the manuscript. YZ and CMW were responsible for analysis and interpretation of the data and revision of the manuscript. JAE was responsible for the conception and design of the study, interpretation of data, and revision of the manuscript. All authors approved the final version of the manuscript. The authors declare no conflicts of interest.

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http://dx.doi.org/10.1016/j.nut.2014.04.006

study based on an animal model in HF showed that a high intake of saturated fat improves survival in HF compared with a high polyunsaturated fatty acid (PUFA) diet or low-fat diet [7], whereas a study on men with a recent coronary event showed that substituting dietary linoleic acid (LA; a polyunsaturated ω-6 fatty acid) in place of saturated fats increased the rates of death from all causes, coronary heart disease (CHD), and cardiovascular disease (CVD) [8]. An updated meta-analysis of LA intervention trials showed no evidence of cardiovascular benefit [8], contrary to the widely accepted advice to substitute polyunsaturated fats for saturated fats for CHD risk reduction. Finally, the GISSI-HF (Gruppo Italiano per lo Studiodella Sopravvivenza nellInfarto-Miocardico–Heart Failure) trial suggested a modest survival advantage from dietary supplementation with ω-3 on mortality and cardiovascular hospital admission in HF [9,10]; however, recent clinical trials and meta-analysis have not shown a clear evidence of benefit from the supplementation with ω-3 on mortality in patients at risk for or with established CVD [11,12].

Little information regarding dietetic management is provided in the major HF management guidelines [1,13–15], principally due to the lack of empirical evidence that can support a recommendation in HF.

The main objective of this study was to evaluate the association of macronutrients (protein, carbohydrates, and fats) and certain micronutrients (sodium, potassium, magnesium, calcium, and iron) with total mortality in patients with chronic HF. To test the association between dietary fats and mortality, intake of total fat was broken down into monounsaturated (MUFAs), PUFAs, and saturated fatty acids (SFAs).

Materials and methods

Study population and methods

This was a secondary analysis of a randomized controlled trial on the effects of sodium restriction in 203 ambulatory HF patients attending the Heart Failure Clinic of the Instituto Nacional de Ciencias Medicas y Nutricion "SZ," in Mexico City, Mexico. The design and methodology of the trial were published elsewhere [16]. Overall, patients were recruited if they were ≥18 y and had a confirmed diagnosis of HF (both reduced and preserved systolic function). They were excluded if they had a specific nutritional regimen, other than general recommendations for a cardiovascular healthy diet (e.g., enteral feeding), renal failure, uncontrolled dysthyroidism, hepatic failure, unstable ischemic heart disease, recent myocardial revascularization procedures (percutaneous and/or surgical revascularization in the previous 3 mo), severe arrhythmias, HF secondary to chemotherapy, suspicion of malignancy, or limb amputations. Patients were randomized to the intervention (sodium- and fluid-restricted diet) or control group (general nutritional recommendations).

For the purpose of this secondary analysis, patients with a complete 3-d food record (containing 3 d recorded) at baseline visit were included and both control and intervention arms were combined.

Dietary intake assessment

Dietary intake was estimated by using a 3-d food record. This dietary assessment tool has been shown to have a higher validity and agreement than a 103-item food frequency questionnaire as compared with 9-d food record [17] and has been previously used in populations with HF [18–21]. Patients were instructed to record all food and beverages consumed during the 3 d before the baseline evaluation, including 2 weekdays and 1 weekend day. Patients also were asked to provide detailed information about each food item, including portion size, use of condiments, and description of food preparation methods (grilled, fried, boiled, etc.). All food records were reviewed by the dietitian during an interview with the patient to clarify food-item descriptions and portion sizes and to identify any missing food items. Food records were analyzed by trained personnel, with a computerized food database (SCVAN 1,0, 1993, Instituto Nacional de Ciencias Medicas y Nutricion SZ, Mexico City, Mexico), which contains the nutritional value of common Mexican foods. Missing foods were added by using the Mexican Food and Nutrient Values Tables [22].

A mean dietary intake from the 3-d recall was estimated for energy, fiber, macronutrients (carbohydrates, protein, and total fat), and micronutrients (sodium, potassium, magnesium, calcium, and iron). Dietary intake of MUFAs, PUFAs, and SFAs was determined. Dietary intake for macronutrients, including different types of dietary fat, was estimated as g/d and percentage of daily energy intake (DEI) as well.

Clinical information

Information regarding anthropometrics, comorbidities, medical treatment, and cardiac function (left ventricular ejection fraction) was included in this analysis.

Primary outcome

All-cause mortality within 12 mo was the primary outcome. Mortality date and cause of death were verified in each case by reviewing the clinical chart and death certificate when available.

The original trial was approved by the Research Ethics Board for biomedical research in humans of the Instituto Nacional de Ciencias Medicas y Nutricion and all patients gave written informed consent to participate in the study.

Statistical analysis

Continuous variables were expressed as median (25th–75th percentiles), and categorical variables were presented as percentages. Kaplan-Meier survival analysis estimated all-cause mortality through the follow-up period in the entire study population.

Associations between each dietary factor and all-cause mortality were individually tested using Cox proportional hazards regression, and are reported as unadjusted and adjusted hazard ratios (HR) with 95% confidence intervals (CI). Restricted cubic spline functions were used to examine the linearity assumption of the dietary factors [23,24]. Dietary factors with $P \leq 0.20$ in the unadjusted individually tested model were included in the full multivariable model, and then stepwise selection ($P \leq 0.20$ as "in" criteria and $P \geq 0.05$ as "out" criteria) was applied to generate the final model. These associations were adjusted for age, sex, body mass index, baseline ejection fraction ≤45%, and randomized intervention.

All statistical tests were two-sided, and were considered statistically significant when $P < 0.05$. Analyses were performed using SAS (version 9.3; Cary, NC, USA).

Results

Of the 203 patients included in the original trial, 118 provided a food record with 3 d recorded during the baseline assessment. Of these 118 patients, 5 were lost in follow-up. Patients in this analysis and the overall trial population were similar in characteristics and outcomes (data not shown). Baseline characteristics for the patients included in this study are presented in Table 1. Most of the population was male; ischemic etiology was present in 49% of the patients; and the majority of the patients were New York Heart Association functional class 1. Baseline dietary intake for macro- and micronutrients is shown in Table 2. Median of intake as percentage of DEI for carbohydrates was 54%; protein, 14.5%; total fat, 31.8%; MUFAs, 9.2%; PUFAs, 5.3%; and SFAs, 8.2%.

The Kaplan-Meier estimate for all-cause mortality rate within 1 y was 14.8%. Unadjusted associations of macro- and micronutrients intake with all-cause mortality individually tested are shown in Figure 1. Of all the dietary factors individually examined, only PUFAs and SFAs were significantly associated with 1-y mortality. The rest of the macronutrients, fiber, or micronutrients (sodium, potassium, magnesium, calcium, and iron) were not associated with 1-y mortality. The full multivariable model included PUFAs and SFAs and was adjusted for age, sex, body mass index, ejection fraction (≤45% cut), and randomized intervention (Fig. 2). In this model, PUFAs and SFAs remained significantly associated with all-cause mortality. For every unit (either g or %DEI) increase per day in PUFAs, the relative survival association ranged from 16% to 33%. Conversely, every unit (either g or %DEI) increase per day in SFAs was associated with a 13% to 15% (relative) excess hazard of mortality within 1 y.