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Dietary inflammatory index and mortality in hemodialysis patients by path analysis approach (NUGE-HD study)

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ABSTRACT

Objective: The aim of this study was to evaluate the interrelationships between dietary, nutritional, and inflammatory factors in predicting all-cause mortality among individuals in hemodialysis (HD) treatment.
Methods: Participating in this study were 137 patients undergoing HD (58.4% men, 61.7 ± 15.4 y of age) from the NUtrition and GEnetics on HemoDialysis outcomes (NUGE-HD study) cohort. Sociodemographic, anthropometric, and clinical data were collected. Dietary inflammatory index scores were calculated from a quantitative food frequency questionnaire. Plasma C-reactive protein was used as an inflammatory marker. Data were analyzed by path analysis.
Results: During the 2-y follow-up, 27 patients (19.7%) died. Compared with survivors, non-survivors were older ($P = 0.01$) and had lower body mass index ($P = 0.04$). In relation to direct (unmediated) associations, dietary inflammatory index ($P = 0.049$) and C-reactive protein ($P = 0.016$) were positively associated, whereas body mass index was negatively associated with mortality ($P = 0.012$). There were no indirect (mediated) associations of the variables evaluated with mortality.
Conclusion: More proinflammatory diet and systemic inflammation have a direct association with mortality among patients undergoing HD therapy. Additionally, more proinflammatory diet is associated with unhealthy dietary pattern.

Introduction

Patients undergoing hemodialysis (HD) treatment experience premature mortality and a life expectancy of an average of 3 to 4 y once they commence dialysis [1]. Despite continuing progress in HD therapy, the mortality rate of patients undergoing maintenance dialysis is still high. Cardiovascular disease (CVD) is the most common cause of death in this population, accounting for about 40% of deaths [1,2]. Chronic systemic inflammation, frequently present in individuals undergoing HD therapy, is an important determinant of the high CVD mortality and overall mortality [3]. C-reactive protein (CRP) is associated with the development of coronary artery disease, even in the absence of dyslipidemia, and is therefore both a predictor of mortality and an independent risk factor for CVD in patients undergoing HD therapy [4,5].

Diet may play a central role in the regulation of chronic inflammation [6] because specific foods and nutrients have a range of anti- or proinflammatory properties that influence immunologic, and inflammatory markers [7]. In this sense, the dietary inflammatory index (DII) has been used to quantify the overall inflammatory potential of diet, based on the pro- and anti-inflammatory

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properties of ≤45 different dietary components [8]. A major advantage of the DII is that the scoring is not dependent on specific population means or recommendations of intake [8].

Previous work has indicated a consistent positive association between the DII score and risk for obesity [9], CVD [10], metabolic syndrome, and overall mortality [11]. Additionally, higher (i.e., more proinflammatory) DII scores, which are a good indicator of unhealthy dietary patterns, are associated with excessive body weight [9,12]. In patients undergoing HD therapy, the DII has shown significant associations with reliable measures of malnutrition and inflammation and is now established as a good tool for assessing the overall inflammatory potential of diet in this population [13]. In another study, also with individuals undergoing HD therapy, the obese group had a greater inflammatory potential in the diet, as assessed by the DII [14].

Until now, the association of DII with mortality among individuals in HD treatment has not been described. Therefore, investigating the quality of the diet, especially in patients undergoing HD therapy who suffer from dietary restrictions, is essential to improve the inflammatory profile, as well as its influence on the high mortality rate. Overall, the aim of the present study was to evaluate the interrelationships between dietary, nutritional, and inflammatory factors in predicting all-cause mortality among individuals undergoing HD therapy.

Materials and methods

Design and study population

One-hundred thirty-seven individuals undergoing HD therapy (80 men and 57 women; mean age, 61.7 ± 15.4 y) from the NUGE-HD (NUtrition and GEnetics on HemoDialysis) outcomes cohort, treated at a dialysis center located in the interior of the state of Minas Gerais in Brazil, participated in this study. Treatment with HD for ≥1 mo and age ≥18 y were the inclusion criteria. Patients with auditory deficiency, newly implanted catheters, and unstable hemodynamics (according to the service protocol) were not included. All participants of this study underwent HD (3–4 h, three times/wk) with blood flow >250 mL/min and dialysate flow of 500 mL/min. Patients received HD therapy for a median of 3.1 y (1.5–6.3 years) when they were included in this study. The causes of chronic kidney disease were hypertensive nephrosclerosis in 54 patients (39.4%), diabetic nephropathy in 47 (34.3%), polycystic kidney disease in 9 (6.6%), chronic glomerulonephritis in 5 (3.6%), and other or unknown etiologies in 22 patients (16.1%). The mean urea clearance divided by the volume of the distribution of urea (Kt/V urea) was 1.6 ± 0.3.

All patients gave informed consent before participating in the study, in accordance with the principles of the Declaration of Helsinki. The Human Research Ethics Committee of the Universidade Federal de Viçosa approved the study.

Food consumption and DII

To estimate food consumption, we used a quantitative food frequency questionnaire (FFQ) adapted for Brazilian people, based on a questionnaire developed for dialysis patients [15] and consumption information collected in a 24-h recall previously applied to the studied population. During routine HD sessions in the dialysis unit, the FFQ was used by trained researchers to interview patients. For patients with any sign of cognitive impairment, the responses were confirmed with caretakers. During the interview, a photographic album [16] with similar portions was used, so that the interviewee chose the categories of food portions from the album, corresponding to the habitual intake.

To calculate dietary intake, consumption of each food item was converted to g/d and the daily consumption of each nutrient was calculated, according to the nutritional composition of Brazilian food tables [17,18] using a Microsoft Excel spreadsheet, especially designed for this.

To calculate DII scores, we used the method developed by Shivappa et al. [8]. The FFQ-derived dietary information was used to calculate DII scores for all participants [8] based on 29 food parameters, as follows: energy, carbohydrate, fat, protein, dietary fiber, ω-3 fatty acids, ω-6 fatty acids, polyunsaturated fatty acids, monounsaturated fatty acids, saturated fatty acids, trans-fatty acids, cholesterol, vitamin A, thiamin, riboflavin, niacin, vitamin B_6, folate, vitamin B_{12}, vitamin C, vitamin D, vitamin E, β-carotene, retinol, magnesium, zinc, iron, selenium, and ethanol. The higher DII score reflects a more proinflammatory diet, whereas more negative values represent a more anti-inflammatory diet. To control for the effect of total energy intake, the DII was calculated per 1000 kcal.

To evaluate the consumption of fresh and minimally processed foods, was used the NOVA classification proposed by Monteiro et al. [19], which categorizes foods according to processing degree. The foods considered for this study as fresh and minimally processed are presented in Supplementary Table 1.

Other variables

Approximately 30 min after the end of the HD session, body weight (kg) and height (cm) were measured using standard techniques, as previously described [20]. Body mass index (BMI; kg/m^2) was then calculated. Non-fasting blood samples were collected before the initiation of dialysis to measure albumin, hemoglobin, high-sensitivity CRP, and urea. Albumin, hemoglobin, and urea were analyzed using routine laboratory techniques from the service itself. We used the Latex CRP Reagent of the Beckman Coulter AU system (Beckman Coulter, Inc.) to determine CRP concentrations. Individuals were categorized according to cardiovascular risk based on following CRP values: high (>3mg/L), medium (1–3 mg/L), and low risk (<1mg/L) [21]. Patients with CRP values >10 mg/L were excluded from the analyzes. Kt/V urea was calculated using the equation proposed by Daugirdas II [22].

Study participants were interviewed using a semistructured questionnaire to obtain sociodemographic (sex, age, and scholarity) and clinical (underlying kidney disease) data. The medical records of each patient also were carefully reviewed, and data related to underlying kidney disease or other comorbidities and time on HD were collected.

Follow-up

Study participants were followed from the date of the first interview, which occurred between September and October of 2017, until death or end of follow-up (September 2019). To obtain information on the outcome, such as death dates and the basic cause of death, medical records were consulted in the HD service to verify whether the patients whose data was collected at the beginning of the study were still alive. These data were then checked in the Brazilian Mortality Information System (http://datasus.saude.gov.br/sistemas-e-aplicativos/eventos-v/sim-sistema-de-informacoes-de-mortalidade), in which information on the basic cause of death (International Classification of Diseases-10) was also stored.

Statistical analysis

Descriptive analyses were performed using the SPSS version 21 (SPSS, Chicago, IL, USA), by calculation of absolute and relative frequency, mean and SD or median and interquartile range, when appropriate. Differences between survivors and non-survivors were analyzed using Student's t test or Mann–Whitney U test, according to normality of quantitative variables. The Pearson's χ^2 test was performed to verify the differences between the two groups of categorical variables.

The MPlus software, version 5, was used to explore the interrelations between variables using path analysis. Path analysis, a subset of structural equation modeling [23], is an extension of regression analysis that simultaneously estimates the linear associations between all variables in a model [24] and allows for evaluating the total, direct, and indirect association of each variable with outcome. Direct associations represent the direct relationships between two variables, that is, those are not mediated by other model variables, and can be interpreted similarly to a regression coefficient. Indirect associations, in turn, express a sequence of paths with at least one intermediate or mediating variable, and are calculated by multiplying the coefficients of direct associations between the variables belonging to that path. Finally, the total association is calculated from the sum of direct and indirect coefficients for the association between two variables [25,26].

Figure 1 depicts the tested theoretical model, where the main response variable is mortality. In this model, all relationships were adjusted for sex, age, and scholarity. Additionally, the associations with mortality were adjusted for Kt/V urea, time on HD, hemoglobin, and albumin. Standardized coefficients with their respective P values were estimated. Odds ratio (OR) values were obtained from the exponentiation of non-standard coefficients. The robust maximum likelihood method was used to estimate the parameters. It is a robust method that does not require the assumption of normal multivariate data distribution [25].

To verify the fit of the model, some measurements were analyzed: root mean square error of approximation (RMSEA) and standardized root mean square residual (SRMR), which are based on model residuals, with values <0.06 indicating that the theoretical model fits the data [27,28]; and Tucker–Lewis index (TLI) and comparative fit index (CFI), where values >0.90 indicate a good fit of the model [25,29].

In all analyzes, $\alpha = 0.05$ was adopted as the level for statistical significance.

Results

At the end of 2 y, 27 patients died (19.7%), with a median follow-up time of 708 d. Five patients (18.5%) died of cardiovascular causes, including cardiac insufficiency, cardiac arrhythmia, and

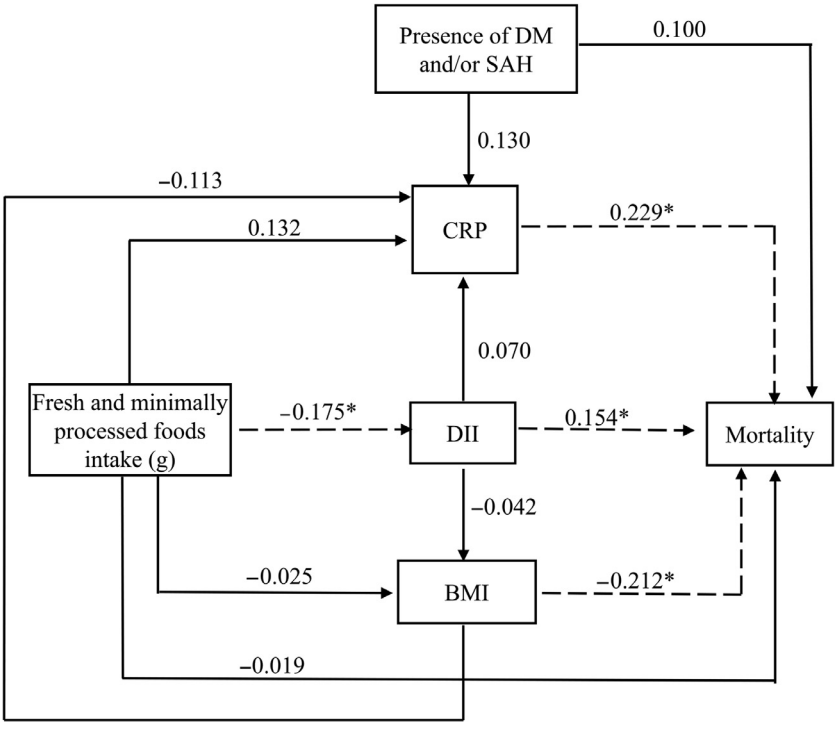

Fig. 1. Path model of relationships between dietary, nutritional, and inflammatory variables and mortality among subjects under HD therapy. In path analysis, all variables were considered continuous, except mortality and presence of DM and/or SAH. In this model, all relationships were adjusted for sex, age, and education. In addition, the associations with mortality were adjusted for Kt/V urea, time on HD, hemoglobin, and albumin. *$P < 0.05$. Dashed lines indicate paths with statistical significance. BMI, body mass index, CRP, C-reactive protein, DII, dietary inflammatory index; DM, diabetes mellitus; HD, hemodialysis; Kt/V urea, mean urea clearance divided by the volume of the distribution of urea; SAH, systemic arterial hypertension.

cardiogenic shock; 4 patients (14.8%) died of diabetes mellitus with kidney complications; 4 patients (14.8%) died of unknown causes; 3 (11.1%) died of hypertensive kidney disease with kidney failure; and 2 (7.4%) died of cancer. Other causes such as sepsis, pneumonitis, and chronic obstructive pulmonary disease were responsible for the other deaths (9 patients; 33.4%).

Table 1 shows the demographic, clinical, nutritional, and food intake characteristics of the survivors and non-survivors. Compared with survivors, non-survivors were older ($P = 0.01$) and had lower BMIs ($P = 0.04$). For the other variables evaluated, no statistically significant difference was observed between the two groups (Table 1).

Figure 1 shows the standardized coefficients for the direct associations of the model. DII ($P\ 0 = 0.049$) and CRP ($P = 0.016$) were positively associated with mortality. BMI was negatively associated with mortality ($P = 0.012$). Finally, an inverse relationship was observed between fresh and minimally processed food intake and DII ($P = 0.049$). Consumption of fresh and minimally processed foods did not have significant direct associations with mortality (Fig. 1).

Table 2 presents ORs for direct, indirect, and total associations of variables with mortality. An increase of 1 unit in the DII score was associated with a 4.4% increase in risk for death from all causes. Similarly, a 1-unit increase in CRP (mg/L) was associated with a 0.6% increase in mortality. On the other hand, the increase of 1 unit in the BMI (kg/m^2) was associated with 2% reduction in the risk for death. No significant indirect association of fresh and minimally processed food intake, DII, and BMI with mortality was identified.

The path model presented satisfactory adjustment indices. The RMSEA was 0.011, SRMR 0.034, TLI 0.979, and CFI 0.993, which suggest a good fit of the model. The total effect size (R^2) of the mortality measure was 19.8%, indicating the hypothesized model explained 19.8% of the variance in mortality.

Discussion

To our knowledge, this was the first study to investigate the association of DII, as well as to identify the interrelationships between dietary, nutritional, and inflammatory factors, with all-cause mortality in individuals undergoing HD therapy. Furthermore, this was the first study to use this methodological approach of path analysis to assess the interrelationships between the variables studied and mortality in this population. Path analysis has stood out as it allows to simultaneously estimate multiple associations between variables, including mediation [24].

The findings of this study show that a more proinflammatory diet (higher DII values) had a direct and positive association with mortality. Some prospective cohort studies with non-dialytic populations have reported increased mortality in participants with the highest DII scores compared with those with the lowest scores, ranging from 8% to 34% [30–32].

A possible explanation for the direct association between DII and mortality is the effect of proinflammatory diet on increasing levels of cytokines such as interleukin (IL)-1 and tumor necrosis factor (TNF) which, in turn, causes attraction and migration of inflammatory cells into vascular tissue [33,34]. These inflammatory markers also increase the expression of cellular adhesion molecules such as selectins and cadherins, which mediate adhesion of white blood cells to the vascular endothelium, increasing CVD risk and related mortality [35]. Besides, the proinflammatory diet, represented by higher DII scores, has been associated with increased risk for CVD and CVD mortality, justifying the direct association observed in the present study between DII and mortality.

Table 1
Sociodemographic, clinical, nutritional, and food intake characteristics of survivors and non-survivors undergoing HD therapy at 2 y of follow-up

Variables	Total (n = 137)	Survivors (n = 110)	Non-survivors (n = 27)	*P*-value
Follow-up (d), Median (IQR)	708 (700–715)	708 (705–715)	312 (147–538)	<0.001
Sex, n (%)				
Men	80 (58.4)	65 (59.1)	15 (55.6)	0.74
Women	57 (41.6)	45 (40.9)	12 (44.4)	
Age, n (%)				
≥60 y	83 (60.6)	61 (55.5)	22 (81.5)	**0.01**
≤59 y	54 (39.4)	49 (44.5)	5 (18.5)	
Scholarity,* n (%)				
Illiterate†/Fundamental incomplete	89 (69)	71 (68.3)	18 (72)	0.69
Fundamental complete‡/Incomplete high school	23 (27.8)	18 (17.3)	5 (20)	
Complete high school§/Superior complete‖ and incomplete	17 (13.2)	15 (14.4)	2 (8)	
Time on HD (y), median (IQR)	3.1 (1.5–6.3)	3.1 (1.2–6.3)	3.1 (1.9–7.5)	0.42
Presence of DM and/or SAH, n (%)				0.082
Yes	101 (73.7)	78 (70.9)	23 (85.2)	
No	36 (26.3)	32 (29.1)	4 (14.8)	
Kt/V urea, mean (± SD)	1.6 ± 0.3	1.6 ± 0.3	1.6 ± 0.3	0.64
BMI (kg/m^2), med (IQR)	23.7 (20.6–26.1)	24 (21.7–26.1)	21.3 (18.6–26.2)	**0.04**
Albumin (g/L), median (IQR)	37 (35–38.5)	38 (35–38.5)	35.8 (32.3–39.1)	0.12
CRP, n (%)				
Low CV risk	17 (13.1)	14 (13.6)	3 (11.1)	0.83
Medium CV risk	33 (25.4)	27 (26.2)	6 (22.2)	
High CV risk	80 (61.5)	62 (60.2)	18 (66.7)	
Hemoglobin (g/L), mean (±SD)	111.5 ± 15.1	111.3 ± 14.8	112.4 ± 16.6	0.76
DII, mean (±SD)¶	−1.4 ± 1.4	−1.4 ± 1.4	−1 ± 1.3	0.33
Fresh and minimally processed foods intake, n (%)†				
>median (760.5 g/d)	50 (49.5)	43 (48.9)	7 (53.8)	0.74
≤median (760.5 g/d)	51 (50.5)	45 (51.1)	6 (46.2)	

BMI, body mass index; CKD, chronic kidney disease; CRP, C-reactive protein; CV, cardiovascular; DII, dietary inflammatory index; DM, diabetes mellitus; HD, hemodialysis; SAH, systemic arterial hypertension; IQR, interquartile range
Pearson's χ^2, Mann–Whitney test (median and IQR) and Student's *t* test (mean and SD). *P*-values in **bold** have statistical significance ($P < 0.05$)
*Scholarity: missing data for nine participants.
†Illiterate: individual who cannot read and write.
‡Fundamental complete: 8 y of education.
§Complete high school: 11 y of education.
‖Superior complete: completed the higher education course.
¶DII and fresh and minimally processed foods intake: missing data for 36 participants.

Table 2
Direct, indirect, and total coefficients of the path analysis

Relationship	Mediators	Associations	Standardized coefficient	*P*-value	OR
Fresh and minimally processed food intake → Mortality		Direct	−0.019	0.85	1.000
	DII	Indirect	−0.026	0.16	1.000
	CRP		0.030	0.37	1.000
	BMI		0.005	0.80	1.000
	DII → BMI		−0.002	0.66	1.000
	DII → CRP		−0.003	0.51	1.000
	BMI → CRP		0.001	0.78	1.000
	DII → BMI → CRP		0.000	0.69	1.000
		Total	0.006	0.96	1.000
DII → Mortality		Direct	0.154	**0.049**	1.044
	BMI	Indirect	0.009	0.64	1.003
	CRP		0.016	0.47	1.005
	BMI → CRP		0.001	0.67	1.000
		Total	0.172	**0.046**	1.052
CRP → Mortality		Direct	0.229	**0.016**	1.006
BMI → Mortality	CRP	Direct	−0.212	**0.012**	0.980
		Indirect	−0.026	0.359	0.998
		Total	−0.238	**0.006**	0.978
Presence of DM and/or SAH → Mortality		Direct	0.100	0.155	1.095
	CRP	Indirect	0.030	0.188	1.027
		Total	0.130	0.074	1.126

BMI, body mass index; CRP, C-reactive protein; DII, dietary inflammatory index; DM, diabetes mellitus; SAH, systemic arterial hypertension
P-value in **bold** has statistical significance ($P < 0.05$).

Therefore, the DII should be used as tool to characterize the inflammatory potential of diet and to predict CVD incidence and mortality [36].

We also observed a direct relationship between CRP, a well-recognized inflammatory marker, and mortality. Chronic low-grade inflammation is common feature in individuals undergoing HD therapy, and the causes are complex and multifactorial. These include the repeated contact of blood mononuclear cells with dialysis tubes and dialyzer membranes, impurities in the dialysis water and/or dialysis solution, oxidative and carbonyl stress, increased release and decreased clearance of inflammatory cytokines, clinical or subclinical infection of the vascular access port, malnutrition, and increased oxidative stress [37]. Inflammatory markers such as TNF and CRP are powerful independent predictors of risk for atherosclerosis, CVD, and mortality in individuals undergoing HD therapy [38]. Evidence suggests that elevated circulatory levels of IL-6 and CRP are strong correlates of mortality in patients undergoing HD therapy [39,40].

In the present study, we did not observe a significant association of DII with CRP concentrations. This may be due to the fact that the causes of an elevated CRP are complex and multifactorial and extend beyond diet. Although the DII is an effective measure of diet-associated inflammation, other pathophysiologic processes also influence chronic systemic inflammation.

As expected, BMI was inversely associated with mortality in the present study. Higher BMI values have been associated with longer survival in patients undergoing HD therapy; this is known as the *obesity paradox* [41]. Some mechanisms may explain the protective effect of high BMI in dialysis patients. First, a high BMI reflects higher energy stores, mainly in the form of adipose tissue. Additionally, patients with higher BMI scores have better appetites [42]. When renal function deteriorates and a uremic milieu develops, well-preserved energy stores become increasingly important as a mortality predictor [43]. Second, uremic toxin production is relatively higher in patients with low body weight [44]. Third, endothelial progenitor cell density is related to obesity [45] when not complicated by diabetes [46], thus this endogenous repair mechanism may be better preserved [47]. Finally, most of the individuals assessed in the present study were elderly and thus, the cutoff point for low weight is more sensitive.

We also found that the consumption of fresh and minimally processed foods was inversely associated with DII. This is consistent with a large body of previous work [11]. The beneficial combinations of phytochemicals, antioxidants, fiber, and monounsaturated fats contributed by legumes, fruits, vegetables, and grains may act together to reduce oxidative stress and inflammatory response, enhance fat oxidation, improve insulin sensitivity, and decrease cardiometabolic risk [6,48,49].

Additionally, these nutrients and foods have anti-inflammatory properties, which reduce the inflammatory potential of the diet and, consequently, DII scores. A study with individuals with cardiometabolic risk reported that the highest DII tercile (the more proinflammatory) was associated with more unhealthy diets (represented by fast food and pasta) [12]; whereas in another study using this same population, a greater adherence to the "Healthy" pattern (characterized by a greater intake of whole grain foods, nuts, fruits and natural juices, milk and dairy products, and a low consumption of margarine, butter, oily sauces, alcohol, coffee, and tea) was associated with a lower cardiometabolic risk [50]. Similarly, a study from Cohort of Universities of Minas Gerais (CUME project) showed that the more proinflammatory diet was associated with other unhealthy lifestyles, including physical inactivity, smoking, and obesogenic diet [9]. Finally, a recent review described a balanced and varied diet, rich in natural foods and poor in processed foods, can reduce the low-grade inflammation and related diseases [7].

Taken together, our results suggest that maintaining adequate nutritional status along with the adoption of healthy dietary pattern, which includes increased intake of healthy and anti-inflammatory dietary components and decreased intake of proinflammatory components, is important for decreasing overall mortality in individuals undergoing HD therapy, as previously demonstrated in other populations and chronic conditions.

This was the first study to test the association between the DII and mortality in patients undergoing HD therapy. Novel use of path analysis also is strength. Also, the present study used the DII, which is a universally applicable scoring system that has been validated in numerous other studies [51]. Despite its strengths, dietary data of this study was collected using a food frequency questionnaire, which requires cognitive skills of the individual to remember the consumption of food items listed during determined time, reflecting more habitual than actual diet [52], although this tool is commonly used in nutritional epidemiology [53]. Another limitation was the variables collection only on the baseline, so changes could not be assessed over time. However, to make longitudinal studies feasible, this is a scientific practice widely used in the literature [54–56].

Conclusion

A more proinflammatory diet and systemic inflammation have a positive direct association and BMI has a negative direct association with HD mortality. Not surprisingly, the consumption of fresh and minimally processed foods is inversely associated with DII, reinforcing its relation of more anti-inflammatory diet with healthier dietary pattern. Altogether, our outcomes demonstrate the importance of constantly assessing the nutritional status and dietary intake of patients under HD therapy to improve diet quality and reduce the high mortality rate still present in this population.

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Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.nut.2021.111239.

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