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Original article

Dietary inflammatory index and its association with renal function and progression of chronic kidney disease

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SUMMARY

Background: While evidence exists for an association between the dietary inflammatory index (DII) and cardiovascular diseases, the relation between DII and chronic kidney disease (CKD) is not known.
Objective: To examine the association between DII, renal function and progression of CKD.
Methods: In this cross-sectional study, dietary data from 221 subjects with diagnosed CKD were collected using a validated food frequency questionnaire. DII was calculated based on overall inflammatory effect scores. Renal function was measured by blood urea nitrogen (BUN) and serum creatinine (Cr) level as well as estimated glomerular filtration rate (eGFR).
Results: Patients in the first tertile of DII consumed higher quantities of vegetables, fruits, dairy, calcium, potassium and phosphorus and lower amounts of grains ($P < 0.01$ for all). We did not detect any significant trend for BUN, Cr and eGFR across tertiles of DII in crude and two adjusted models. In a fully adjusted model, an increased risk of being in the higher stage of CKD was found among those in the top tertiles of DII (odds ratio: 2.12; 95% CI: 1.05, 4.26; P for trend = 0.03).
Conclusion: We observed that compliance with a pro-inflammatory diet in patients with CKD may be associated with disease progression.

1. Introduction

Chronic kidney disease (CKD) is referred to heterogeneous conditions in which kidney structure become abnormal and its function has been gradually lost [1]. Although CKD had been in 27th place of the causes of global mortality in 1990, it was in 18th place in 2010 [2]. Global statistics showed that the prevalence of CKD in 2013 was 8–16% [2]. However, local reports revealed that it was more prevalent among Iranian population [3]. Evidence shows that onset and progression of CKD is associated with a chronic pro-inflammatory state [4]. Also, the importance of dietary intake in the context of CKD was addressed by previous study [5].

Dietary inflammatory index (DII) is a literature based scoring system in which both the role of pro- and anti-inflammatory dietary items are considered [6]. Previous studies reported that several foods and nutrients such as whole grains, fruits, vegetables, fish, vitamin E, vitamin C and magnesium had an anti-inflammatory effect [7–9]. In contrast, refined grains, red meat, high-fat dairy products and simple sugar were related to increased inflammatory markers [10]. These dietary components are used in DII calculation. A benefit of DII is that it represents overall diet

Abbreviations: ANOVA, analysis of variance; BUN, blood urea nitrogen; CKD, chronic kidney disease; Cr, creatinine; CRP, C-reactive protein; DII, dietary inflammatory index; eGFR, estimated glomerular filtration rate; FFQ, food frequency questionnaire; IL-6, interleukin-6; MDRD, Modification of Diet in Renal Disease; OR, Odds ratio.

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rather than individual nutrients and foods [11]. Moreover, this scoring system is dependent on findings of scientific publications rather than population means or recommended intakes [11]. There was an inverse association between DII and C-reactive protein concentration in adults [12]. Also, the DII was directly related to risk of colorectal cancer and longer hospitalization in colorectal cancer patients [6,13]. There was an inverse association between DII and lung function in patients with asthma [11]. Moreover, subjects with higher DII had a greater risk of metabolic syndrome [14]. As onset and progression of CKD is associated with a chronic pro-inflammatory state [5], we hypothesized that DII may be related to kidney function and progression of CKD. Therefore, the aim of this was to evaluate the association between DII and markers of kidney function and progression of CKD.

2. Methods

This study was based on the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) [15]. This article is a part of larger study in which we assessed the association between dietary factors and blood urea nitrogen in patients with CKD. Sample size in original study was calculated by following formula: $n = [Z_{1-\alpha/2}+Z_{1-\beta}]^2/[0.5 \times \log(1 + r/1-r)]$ According to this formula ($r = 0.2$, $\alpha = 0.05$ and $\beta = 0.20$), 193 patients were recruited for the study.

A professional staff calculated estimated glomerular filtration rate (eGFR) using Modification of Diet in Renal Disease (MDRD) equation for all subjects who referred to nephrology clinics [16]. Finally, we enrolled 211 subjects with CKD (eGFR < 60 mL/min/1.73m2) for this cross-sectional study [17]. CKD was classified based on the following criteria: $30 \leq$ eGFR $\leq$59 mL/min/1.73 m^2: stage 3; $15 \leq$ eGFR $\leq$29 mL/min/1.73 m^2: stage 4; eGFR <15 mL/min/1.73 m^2: stage 5 [17]. Other diseases (e.g., diabetes and nephrolithiasis) had been controlled by related specialists. Written consent was signed by each patient. This cross-sectional study was conducted in Isfahan, Iran in 2015. The Ethic Committee of [removed for blind peer review] approved the study protocol (Code: [removed for blind peer review]).

Food intake of individuals during the previous year was assessed using an interviewer-administered food frequency questionnaire (FFQ). This semi-quantitative FFQ contained 168 food items. Previous studies showed acceptable validity and reliability coefficient for used FFQ [10,18]. Nutritionist IV (N-Squared Computing, Salem, OR) software was used to analyze composition of consumed foods. Under- or over-reported data (<800 or >4200 kcal/d) were removed.

We used the method introduced by Shivappa et al. to calculate DII [19]. Data regarding of 29 dietary items from total of 45 were collected in the present study: energy, carbohydrate, fat, protein, dietary fiber, n-3 fatty acids, n-6 fatty acids, poly-unsaturated fatty acids, mono-unsaturated 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, magnesium, zinc, iron, selenium and onion, garlic. DII score was calculated using adjusted scores published by Shivappa et al. [19]. Overall inflammatory effect scores were multiplied by the intake for each patient. Then each dietary item score product was summed. Calculated scores were divided by 100 to make statistical analyses easier.

A blood sample was taken after subjects were on overnight fasting for 12 h. Samples were centrifuged at 3000 × g for 10 min. Serum creatinine (Cr) was determined by colorimetric reflectance spectrophotometry. All kits were produced by Pars Azmoon Inc.

Information was orally asked on demographic characteristics. We determined socioeconomic status on the basis of income, occupation, education and region of residence. Body weight was recorded in lightweight clothing and after removal of shoes by digital scale to the nearest 0.1 kg. Height was self reported. Participants were asked to report sedentary time and activities to measure physical activity [20]. Seated systolic and diastolic blood pressure was recorded using a standard mercury sphygmomanometer.

Normality of variables distribution was checked using Kolmogorov–Smirnov test. As we used an interviewer administered questionnaire, we had no missing data. Chi-square test and analysis of variance (ANOVA) were performed to compare nominal, ordinal and continuous variables by tertiles of DII. Dietary intakes were adjusted for total daily energy intake using a residual method. DII has no determined cutoff point. Therefore, reported dependent variables across tertiles of DII. As gender is a variable in calculation of eGFR [16], we did not include it to adjusted models for eGFR and risk of CKD progression. Odds ratio (OR) and 95% CI was calculated using logistic regression. ORs were reported in two adjusted models. Confounding variables in the first adjusted model were socioeconomic status, height and weight. Further adjustment was applied for systolic and diastolic blood pressure in second adjusted model. Continuous variables were reported as mean ± SD. We used SPSS version 20 (IBM) to analyze data.

3. Results

From 778 subjects who were screened, 221 individuals were eligible for inclusion in study. As we used an interviewer administered questionnaire, we had no missing data. Table 1 demonstrates general characteristics of subjects with CKD across tertiles of DII. The percent of males was insignificantly higher in top tertile ($P = 0.42$). Similarly, there was no significant difference in other variables across tertiles of DII.

Energy adjusted intake of food groups and nutrients across the tertiles of DII is shown in Table 1. Patients in the lowest tertile of DII consumed more amounts of vegetables, fruits, dairy, calcium, potassium and phosphorus ($P < 0.01$ for all). Moreover, intake of grains was significantly higher in the top tertile of DII ($P < 0.01$). Also, a marginally significant higher meat consumption was observed among those in the first tertile of DII in compared with the last tertile ($P = 0.06$). There was no significant difference in intake of nuts ($P = 0.10$), sugar ($P = 0.25$) and oils ($P = 0.36$) across tertiles of DII.

Table 2 displays mean of renal function variables across tertiles of DII. We could not detect any significant trend for BUN, Cr and eGFR across tertiles of DII in crude and two adjusted models.

We show odds ratios for being in the higher stage of CKD according to tertiles of DII in Table 3.

In the crude model, an increased risk of being in the higher stage of CKD was found among those in the top tertiles of DII (P for trend = 0.03). Similar finding was also observed in multivariate adjusted model (P for trend = 0.03).

4. Discussion

The results of our study showed that patients in the highest tertiles of DII had increased risk of being in the higher stage of CKD. To the best of our knowledge, this is the first study which examined association between DII and risk of progression of CKD. Furthermore, the intake of food groups and nutrients was significantly different across tertiles of DII.

As shown in the result section, patients in the last tertile of DII consumed more amounts of grains. Similar to our finding, an observational study conducted in Italy showed that subjects in the top quartile of DII consumed larger amounts of bread [21]. In our

Table 1
General characteristics and dietary intakes of patients with chronic kidney disease across tertiles of dietary inflammatory index.

Variables	Tertiles of dietary inflammatory index			P[b]
	T1 (≤−4.55) (n = 73)	T2 (−4.54, −1.14) (n = 74)	T3 (≥−1.13) (n = 74)	
Age (year)	58.42±13.53[a]	57.62 ± 16.47	53.69 ± 15.28	0.13
Male (%)	64.4	68.9	74.3	0.42
Weight (kg)	73.08 ± 14.62	72.44 ± 11.07	73.99 ± 14.25	0.78
BMI (kg/m^2)	25.80 ± 4.44	25.70 ± 3.98	26.02 ± 4.64	0.91
Physical activity (%)				
Low	54.8	64.9	66.2	
Moderate	39.7	32.4	32.4	
High	5.5	2.7	1.4	0.44
Socioeconomic status (%)				
Low	12.3	17.6	24.3	
Middle	65.8	70.3	60.8	
High	21.9	12.2	14.9	0.22
CKD stage (%)				
Stage 3	69.9	67.6	63.5	
Stage 4	28.8	31.1	35.1	
Stage 5	0.5	0.5	0.5	0.95
Primary renal disease (%)				
Diabetes	24.7	25.7	20.3	
Hypertension	16.4	12.2	20.3	0.77
Diabetes + Hypertension	27.4	33.8	25.7	
Other	31.5	28.4	33.8	
Dietary intakes[c]				
Grains (g)	296.74 ± 94.86	329.12 ± 112.05	387.57 ± 97.79	<0.01
Vegetables (g)	322.32 ± 155.28	255.64 ± 116.57	173.01 ± 88.30	<0.01
Fruits (g)	287.62 ± 129.54	248.98 ± 104.57	187.03 ± 96.17	<0.01
Legumes (g)	30.93 ± 24.64	26.50 ± 18.29	26.61 ± 19.50	0.35
Dairy (g)	449.40 ± 285.33	393.32 ± 200.37	328.08 ± 199.90	<0.01
Meats (g)	31.42 ± 17.70	26.69 ± 13.91	25.94 ± 13.62	0.06
Nuts (g)	11.26 ± 6.26	9.51 ± 4.96	9.24 ± 6.89	0.10
Oils (g)	22.81 ± 7.45	21.16 ± 7.23	22.04 ± 6.15	0.36
Sugar (g)	53.71 ± 31.12	55.39 ± 36.72	64.83 ± 58.13	0.25
Calcium (mg)	898.14 ± 345.16	801.61 ± 256.50	710.46 ± 264.25	<0.01
Potassium (mg)	2688.32 ± 799.65	2312.09 ± 598.02	1871.98 ± 461.58	<0.01
Phosphorus (mg)	926.86 ± 295.85	818.88 ± 207.35	745.57 ± 288.72	<0.01

BMI: body mass index, CKD: chronic kidney disease.
[a] Mean ± SD.
[b] Calculated by Chi-square and analysis of variance for qualitative and quantitative variables, respectively.
[c] All values have been adjusted for total energy intake using a residual method.

Table 2
Mean of renal function variables reported by tertiles of dietary inflammatory index among patients with chronic kidney disease.

Variables	Tertiles of dietary inflammatory index			P[b]
	T1 (≤−4.55) (n = 73)	T2 (−4.54, −1.14) (n = 74)	T3 (≥−1.13) (n = 74)	
BUN (mg/dl)				
Crude	30.61±13.26[a]	30.83 ± 13.45	30.26 ± 13.81	0.96
Model 1[c]	30.49 ± 13.40	30.63 ± 13.27	30.58 ± 13.41	0.99
Model 2[d]	30.68 ± 13.03	30.98 ± 12.86	30.04 ± 13.10	0.91
Creatinine (mg/dl)				
Crude	1.93 ± 0.65	2.02 ± 0.77	2.11 ± 0.75	0.34
Model 1	1.96 ± 0.72	2.03 ± 0.71	2.09 ± 0.72	0.57
Model 2	1.97 ± 0.71	2.03 ± 0.71	2.07 ± 0.71	0.68
eGFR (mL/min/1.73m^2)				
Crude	38.19 ± 11.98	38.15 ± 12.92	37.16 ± 12.69	0.85
Model 1	38.64 ± 12.36	38.28 ± 12.25	38.35 ± 12.38	0.56
Model 2	38.49 ± 12.23	38.27 ± 12.15	36.74 ± 12.30	0.65

eGFR: estimated glomerular filtration rate, BUN: blood urea nitrogen.
[a] Mean ± SD.
[b] Calculated by multivariate analysis of variance (in crude model) and multivariate analysis of covariance (in adjusted model).
[c] Model 1: Adjusted for age, sex, socioeconomic status, height and weight.
[d] Model 2: Model 1 + systolic and diastolic blood pressure.

Table 3
Odds ratios and 95% confidence intervals for being in the higher stage of chronic kidney disease according to tertiles of dietary inflammatory index among patients with chronic kidney disease.

Models	Tertiles of dietary inflammatory index			P value[a]
	T1 (≤−4.55) (n = 73)	T2 (−4.54, −1.14) (n = 74)	T3 (≥−1.13) (n = 74)	
Crude	1	0.85 (0.41, 1.77)	1.99 (1.01, 3.95)	0.03
Model 1[b]	1	0.91 (0.44, 1.91)	2.11 (1.05, 4.23)	0.03
Model 2[c]	1	0.91 (0.44, 1.91)	2.12 (1.05, 4.26)	0.03

[a] P-value was calculated by logistic regression.
[b] Model 1: Adjusted for socioeconomic status, height and weight.
[c] Model 2: Model 1 + systolic and diastolic blood pressure.

population, 84% of participants did not consume any type of whole grain products and their grain intake was fully obtained from refined sources (data not shown). Previous studies revealed that consumption of refined grains was associated with higher concentration of inflammatory markers [22,23]. Therefore, it seems that this finding will be repeated in populations who consume high amounts of refined grains.

We found that subjects in the first tertile of DII consumed higher amounts of fruits and vegetables. There was a negative association between inflammation and intake of fruits and vegetables [24]. Fruits and vegetables are rich sources of several nutrients (e.g., folate, dietary fiber and β-carotene) which have negative overall inflammatory effect scores in calculation of DII (18). Therefore, higher intakes of fruits and vegetables were expectable among those with lower DII.

We observed that dairy intake in the first tertile of DII was significantly higher in compared with top tertile. According to our analysis, 77% of patients consumed low amounts (<10%) of low-fat dairy and their dairy intake was mainly obtained from high-fat sources (data not shown). Therefore, the consumption of low-fat dairy was high in our population. Previous study indicated that adherence to a diet restricted in high-fat dairy resulted in lower concentration of inflammatory markers [25].

The results displayed marginally significant higher meat consumption among those in the first tertile of DII in compared with the last tertile. Previous studies reported that red meat intake resulted in higher concentration of inflammatory markers [18,26]. In contrast, evidence showed that fish has an anti-inflammatory effect [27]. Therefore, the type of meat has an important role in concentration of inflammatory markers. In the present study, 83% of participants did not consume large amount of red meat (<50% of total consumed meat) and their meat intake was mainly obtained from white sources (data not shown). Therefore, we observed higher meat intake among those in the lower tertile of DII.

Patients in the lowest tertile of DII consumed more amounts of calcium. It seems that higher calcium intake may be due to more dairy intake in the lowest tertile of DII. Calcium balance study showed that adherence to a diet containing 800 mg calcium resulted in slightly negative to neutral calcium balance [28]. As patients in the last tertile of DII got <800 mg/day calcium, a diet rich in pro-inflammatory dietary items may lead to negative calcium balance.

Findings showed that patients in the lowest tertile of DII consumed more amounts of potassium and phosphorus. It seems that higher potassium and phosphorus intake may be due to more intake of dairy, fruits and vegetables in the lowest tertile of DII. Nutritional recommendations for patients with CKD declared that phosphorus and potassium intake should be <1000 and <2400 mg/d, respectively [29]. Mean of phosphorus intake in the first tertile of DII (926.86 mg/d) was lower than recommended level. However, subjects in the lowers tertiles of DII consumed more than 2400 mg/d of potassium. Therefore, patients with CKD should be aware regarding high-potassium fruits and vegetables to control daily potassium intake.

Patients with CKD are recommended to reduce intake of dietary sources of protein, phosphorus, potassium and sodium [29]. According to DII calculation method [19], dietary protein is considered as a pro-inflammatory nutrient. Phosphorus is not directly included in DII calculation. However, plant-based phosphorus may have anti-inflammatory role because coexisting nutrients such as fiber and flavonoid have negative coefficient in DII calculation [19]. In contrast, coexisting nutrients with animal-based phosphorus (e.g., saturated fatty acid and iron) are classified as pro-inflammatory components [19]. Although dietary potassium is not listed as an item of DII, coexisting nutrients with potassium such as vitamin C and flavonols are defined as anti-inflammatory items in DII calculation.

In the fully adjusted model, an increased risk of being in the higher stage of CKD was found among those in the top tertiles of DII. A recent study shows that inflammation has a remarkable role in progression of CKD [4]. C-reactive protein (CRP) and interleukin-6 (IL-6) are two of inflammatory markers used in calculation of DII [19]. Evidence showed that CRP was a major risk factor and a predictor of cardiovascular diseases in patients with CKD [30,31]. Higher CRP and IL-6 was related to the lower creatinine clearance in predialytic patients [32]. Also, previous study reported that CRP is a predictor of renal function loss [33]. Moreover, elevated level of CRP and IL-6 has been directly associated with all-cause mortality in CKD [34,35]. Stuveling et al. reported that the association between CRP and renal function loss is mediated by body weight. However, they observed an elevated risk for diminished filtration in top quartiles of CRP after adjusting for body mass index [33]. In our study, further adjustment for weight and height did not attenuate observed elevated risk of being in the higher stage of CKD in top tertiles of DII. Therefore, it seems that CRP is independently associated with kidney function. As CRP is an important atherosclerogenic agent [36], its remarkable role in arteriolosclerosis in the renal vasculature should not be neglected. It is possible that loss of renal function lead to lower CRP excretion in urine, more elevated plasma CRP and finally greater arteriolosclerosis in the renal vasculature [33].

Several limitations should be considered in the interpretation of results of the present study. As the information of all patients with CKD was not systematic registered in Iran, we could not use a random sampling method. Therefore, findings of the present cannot be generalized to all patients with CKD. We calculated DII based on 29 dietary items and data regarding 16 dietary items were not available in this study. As the design of the present study was cross-sectional, we could not test the causal relationship between DII and progression of CKD. As patients with CKD have heterogeneous medical conditions, our findings should be tested in specific subgroups of CKD. Information regarding primary renal disease was recorded. However, we could not run sub-analysis because the limited number of subjects in each group affected the validity of statistical method. Although we assessed the relation between BUN, Cr and DII, more biomarkers should be used in future studies.

Using DII is one the strengths of present study. DII looked at overall diet rather than individual nutrients and foods. Moreover, this scoring system is dependent on findings of scientific publications rather than population means or recommendations of intake.

In conclusion, compliance with a pro-inflammatory diet in patients with CKD may be associated with disease progression.

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Authorship

L.A, A.E, A.F and M.M.N designed the study. M.H.R and M.M.N collected data. M.H.R, L.A and A.F performed statistical analysis. M.H.R and P.J.S wrote manuscript.

Conflict of interest

The authors declare that there are no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.clnesp.2018.09.001.

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