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The impact of low dietary inflammatory index diet on clinical parameters in patients with chronic kidney disease: a retrospective comparative study

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Abstract

Background Chronic kidney disease (CKD) poses a significant global health challenge. Inflammation plays a central role in the pathogenesis and progression of CKD, which has been proved to be affected by dietary patterns. To understand how dietary inflammatory index (DII) impacts the disease course and clinical parameters, we aim to explore the relationship between DII and multiple clinical parameters in a specific cohort of CKD patients, and to provide insights into the potential of dietary for managing CKD.

Methods This retrospective comparative study included 145 CKD patients, categorized into a low DII group ($n=77$) and a high DII group ($n=68$) based on their DII values. Clinical data, including demographic characteristics, laboratory parameters, dietary intake, inflammatory markers, renal function, and adverse events, were collected and compared between the two groups.

Results The demographic characteristics were comparable between the groups. The low DII group had significantly lower serum creatinine, phosphorus, and potassium levels ($P<0.05$) and higher hemoglobin levels compared to the high DII group. Protein intake was significantly higher in the high DII group ($P<0.001$), while fiber intake was significantly higher in the low DII group ($P=0.022$). Inflammatory markers, including CRP, TNF-α, fibrinogen, procalcitonin, and WBC, were significantly lower in the low DII group ($P<0.05$). The low DII group also showed better renal function, as indicated by higher GFR and lower urinary albumin excretion ($P<0.05$). Correlation analysis revealed significant relationships between protein intake and inflammation markers (CRP, TNF-α, fibrinogen) and a negative correlation with GFR. Regression analysis confirmed that DII was independently associated with CRP, GFR, and urinary albumin excretion, while protein intake remained significantly correlated with these outcomes.

Conclusion A low DII diet may be associated with improved clinical parameters, inflammatory markers, and renal function in CKD patients. Tailored nutritional strategies focusing on modulating inflammatory status through low DII diets may offer promising avenues for improving renal function, mitigating inflammation, and enhancing overall well-being in individuals with CKD.

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Keywords Dietary inflammatory index, Renal function, Chronic kidney disease, Inflammatory markers

Introduction

Chronic kidney disease (CKD) represents a significant global health burden, characterized by progressive deterioration of renal function and associated with a heightened risk of adverse outcomes, including cardiovascular events, diminished quality of life, and increased mortality [1–3]. CKD affects approximately 10% of the global population and is associated with substantial morbidity and mortality. The impact of CKD extends beyond renal complications, with a range of systemic consequences such as cardiovascular disease, anemia, bone disorders, and metabolic disturbances [4–6]. Furthermore, CKD places a considerable economic burden on healthcare systems worldwide, stemming from the need for complex and prolonged medical care, including dialysis and transplantation [7, 8].

Inflammation is recognized as a key contributor to the pathogenesis and progression of CKD, influencing not only renal function but also contributing to the development of various comorbidities [9–11]. The inflammatory response is intricately involved in the initiation and perpetuation of renal injury, leading to fibrosis, impaired vascular function, and progressive loss of nephrons [12–15].

Dietary patterns play a pivotal role in modulating the inflammatory status of individuals, with evidence suggesting that dietary components can exert pro-inflammatory or anti-inflammatory effects, thereby influencing the overall inflammatory milieu within the body [16–18]. A growing body of research has underscored the impact of diet on inflammatory pathways, with certain dietary components, such as saturated fats, refined sugars, and processed foods, showcasing pro-inflammatory properties that can contribute to a systemic low-grade inflammatory state [19]. Conversely, diets rich in fruits, vegetables, whole grains, and healthy fats have been associated with anti-inflammatory effects, conferring protection against chronic inflammatory conditions [20]. In this context, previous studies have developed the Dietary Inflammatory Index (DII) to assess the total inflammatory potential of diet [21]. DII is a literature based, population-based scoring system aimed at evaluating the inflammatory potential of diet [21]. If the DII value is positive, it indicates that the diet contains pro-inflammatory ingredients; if the DII value is negative, it means that the diet contains anti-inflammatory ingredients. A higher score indicates a stronger pro-inflammatory effect, while a lower score indicates a stronger anti-inflammatory effect [22].

The intricate relationships between dietary choices, inflammatory processes, and health outcomes have prompted extensive investigations aimed at elucidating the potential of dietary in modulating inflammatory status and mitigating the risk of chronic diseases, including CKD. As our understanding of the dietary modulation of inflammation continues to evolve, there is a mounting interest in deciphering the specific impact of DII on the pathophysiology and management of CKD. By exploring the interplay between DII and clinical parameters in CKD patients, there exists the potential to uncover novel avenues for tailored nutritional strategies that may alleviate inflammation and optimize clinical parameters in this population.

Inflammation has been recognized as a key contributor to the pathogenesis and progression of CKD, influencing renal function and contributing to the development of various comorbidities [23]. Despite the prevalence of cross-sectional DII studies in CKD, primarily using NHANES data [24, 25], our study adds to the field by focusing on a specific cohort of CKD patients and providing detailed analysis of the impact of DII on multiple clinical parameters. Clinical outcomes in this study encompass a broad spectrum of clinical parameters, including laboratory values, renal function, and adverse events, collectively aiming to assess the overall impact of DII on CKD patients. The intricate relationships between dietary choices, inflammatory processes, and health outcomes have prompted extensive investigations aimed at elucidating the potential of dietary in modulating inflammatory status and mitigating the risk of chronic diseases, including CKD.

Materials and methods

Inclusion and exclusion criteria

Inclusion Criteria: [1] Adult patients aged 18 years or older; [2] Diagnosis of CKD based on established clinical and laboratory criteria [26].

Exclusion Criteria: [1] Patients with missing or incomplete data in their medical records; [2] Individuals with a diagnosis of acute kidney injury, end-stage renal disease, or those receiving renal replacement therapy; [3] Patients with comorbid conditions or factors that could significantly impact inflammatory status and dietary intake, such as severe infections, malignancies, and major surgical procedures; [4] Participants with a history of significant dietary modifications that could confound the assessment of DII; [5] Patients with COVID-19.

Informed consent was waived in this retrospective cohort study because it exclusively used de-identified patient data that had already been collected as part of routine clinical practice. Since the data did not include any personal identifiers and no direct interaction with patients occurred during the study, there was no risk to patient confidentiality or care.

Data collection

Clinical data of 145 CKD patients treated at our institution from January 2021 to December 2023 were included for analysis. Patients were categorized into a low dietary inflammation index group (Low DII group, $n = 77$) and a high dietary inflammation index group (High DII group, $n = 68$) based on their DII values. The Low DII group comprised 38 males and 39 females, while the High DII group included 34 males and 34 females. The data collected are cross-sectional, reflecting the baseline characteristics of the patients at the start of the study period. The duration of observation for adverse events was from the date of enrollment until the last recorded visit or the end of the study period. Since this is a retrospective study, it was not registered due to the nature of the study design.

Calculation of dii and grouping criteria

The food frequency questionnaire (FFQ) was utilized to assess the dietary intake of the participants, the questionnaire has been previously published and validated in a similar context [27]. Trained investigators surveyed the types and quantities of food consumed by the participants over the previous three months. The food intake was quantified using household measures, food models, or food atlases [28]. The intake of a specific food or nutrient was calculated based on the Chinese Food Composition Table (6th edition) [29]. The overall DII for each individual was calculated based on the average daily nutrient intake [30]. This algorithm incorporates dietary data from 11 representative populations worldwide and assigns DII scores to 45 food parameters based on their potential to influence serum inflammatory markers [31]. A negative DII denotes anti-inflammatory potential, while a positive DII signifies pro-inflammatory potential. To standardize the scores (Z-scores), the daily intake of each dietary component or nutrient was compared to the global mean intake and standard deviation. To minimize the impact of outliers and right skewness, Z-scores were transformed into percentile values. To achieve a symmetrical distribution with 0 at the center, and − 1 (maximum anti-inflammatory) and + 1 (maximum pro-inflammatory) as boundaries, each percentile was doubled, then subtracted by 1, and multiplied by the respective dietary inflammatory effect score [32]. The sum of all the food inflammatory effect scores yielded the DII for each participant. Participants with $DII < 0$ were classified into the Low DII group, while those with $DII > 0$ were classified into the High DII group (participants with $DII = 0$ were not included in the study).

Data collection and variables

Demographic, clinical, laboratory, dietary intake, inflammatory markers, renal function, and adverse event data were collected from the participants' medical records. Demographic characteristics such as age, gender, body mass index (BMI), comorbidities including diabetes mellitus, hypertension, smoking history, and previous cardiovascular events, as well as the duration of CKD were recorded. Laboratory parameters, including serum creatinine, hemoglobin, albumin, phosphorus, calcium, potassium, and parathyroid hormone levels, were measured in our institution's laboratory using standard laboratory techniques. These parameters were assessed through blood samples, with serum or plasma samples used for analysis. All laboratory tests were performed following routine procedures in our accredited clinical laboratories. Commercial kits for the measurement of specific markers (such as serum creatinine and hemoglobin) were employed to ensure consistency and accuracy of results, these data are all recorded in the medical records. Dietary intake data comprising total calories, protein intake, sodium intake, fiber intake, and fluid intake (defined as the total amount of liquids consumed daily, including water, beverages, and other liquid foods) were gathered. Additionally, inflammatory markers, including C-reactive protein, interleukin-6, tumor necrosis factor-α, fibrinogen, erythrocyte sedimentation rate, procalcitonin, and white blood cell count, were assessed using standard laboratory assays in our institution's laboratory. Renal function parameters such as glomerular filtration rate and urinary albumin excretion were also measured from routine clinical tests, and recorded in the medical record. The occurrence of adverse events was documented from patient medical records and follow-up visits.

Statistical analysis

Descriptive statistics were used to summarize the demographic characteristics, clinical parameters, dietary intake, inflammatory markers, renal function, and adverse events. Continuous variables were presented as means with standard deviations, and categorical variables were expressed as frequencies and percentages. The independent samples t-test was employed to compare continuous variables between the two groups, while the chi-square test was utilized for categorical variables. Correlation analysis was performed to assess the relationships between DII and various clinical parameters, including inflammatory markers, renal function, and dietary intake. Pearson's correlation coefficient was used

Table 1 Demographic characteristics of patients in the two groups

Parameters	Low DII group	High DII group	t/χ^2	*P* Value
Age (years)	55.23 ± 6.43	56.89 ± 7.11	1.463	0.146
Gender (M/F)	38 / 39	34 / 34	0	1
Body Mass Index (kg/m^2)	25.07 ± 2.96	24.96 ± 3.59	0.217	0.829
Diabetes Mellitus (%)	15 (19.48%)	18 (26.47%)	0.645	0.422
Hypertension (%)	29 (37.66%)	30 (44.12%)	0.385	0.535
Smoking History (%)	15 (19.48%)	16 (23.53%)	0.152	0.696
Previous Cardiovascular Events (%)	11 (14.29%)	13 (19.12%)	0.311	0.577
Duration of CKD (years)	6.94 ± 1.34	7.18 ± 2.28	0.759	0.449

Note: M, male; F, female; DII, dietary inflammatory index

Table 2 Laboratory parameters indexes of the two groups

Parameters	Low DII group	High DII group	t	*P* Value
Serum Creatinine (mg/dL)	2.89 ± 0.57	3.11 ± 0.59	2.306	0.023
Hemoglobin (g/dL)	11.83 ± 1.56	11.27 ± 1.77	2.021	0.045
Albumin (g/dL)	4.25 ± 0.33	3.97 ± 1.44	1.577	0.119
Phosphorus (mg/dL)	3.57 ± 0.69	3.86 ± 0.78	2.359	0.020
Calcium (mg/dL)	9.24 ± 0.47	9.25 ± 0.53	0.122	0.903
Potassium (mEq/L)	4.28 ± 0.39	4.43 ± 0.46	2.001	0.047
Parathyroid Hormone (pg/mL)	65.47 ± 12.45	68.97 ± 15.64	1.478	0.142

Note: DII, dietary inflammatory index

for continuous variables. Univariate regression analysis was conducted to evaluate the association between DII and clinical outcomes. The regression coefficients and corresponding P values were calculated to determine the strength and significance of these relationships. Multivariate regression analysis was then performed to examine the independent associations of DII with clinical parameters, adjusting for potential confounding factors. Variables that showed significant associations with DII in univariate analysis were included in the multivariate model to assess their independent effects on inflammatory markers and renal function. A *P* value of less than 0.05 was considered statistically significant. All analyses were conducted using SPSS 25.0.

Results

Demographic characteristic

The demographic characteristics of the patients in the low dietary inflammation index group and the high dietary inflammation index group were compared to assess any notable differences (Table 1). The mean age of the low dietary inflammation index group slightly lower than that of the high dietary inflammation index group ($P = 0.146$). Gender distribution was similar between the two groups with no significant difference observed ($P = 1$). The BMI of the two groups also exhibited no significant variance ($P = 0.829$). The prevalence of diabetes mellitus, hypertension, smoking history, and previous cardiovascular events showed comparable percentages between the two groups, with no statistically significant differences found ($P > 0.05$). Moreover, the duration of CKD was akin in both groups ($P = 0.449$).

The above results indicate comparability between the two groups, laying the foundation for subsequent studies regarding the impact of low inflammation index diet on clinical parameters in patients with CKD.

Laboratory parameters

Serum creatinine levels, phosphorus levels and potassium levels were significantly lower in the low dietary inflammation index group compared to the high dietary inflammation index group ($P = 0.023$, $P = 0.02$, $P = 0.047$, respectively). Furthermore, hemoglobin levels were found to be significantly higher in the low dietary inflammation index group than in the high dietary inflammation index group with a *P* value of 0.045 (Table 2). However, no significant differences were observed in albumin, calcium, and parathyroid hormone levels between the two groups ($P > 0.05$).

Dietary intake

Next, we compared the dietary intake of the low and high dietary inflammation index groups and found several significant differences (Table 3). The protein intake was significantly higher in the high dietary inflammation index group compared to the low dietary inflammation index group with a *P* value of less than 0.001. Additionally, fiber intake was significantly higher in the low dietary inflammation index group compared to the high dietary inflammation index group with a *P* value of 0.022. However, there were no significant differences observed in total

Table 3 Comparison of dietary intake between the two groups

Parameters	Low DII group	High DII group	t	*P* Value
Total Calories (kcal/day)	2011.23 ± 301.54	2001.19 ± 356.47	0.182	0.856
Protein Intake (g/day)	70.11 ± 7.29	75.8 ± 8.68	4.245	< 0.001
Sodium Intake (mg/day)	2004.57 ± 403.75	2057.34 ± 349.78	0.843	0.4
Fiber Intake (g/day)	25.69 ± 5.69	23.69 ± 4.68	2.319	0.022
Fluid Intake (mL/day)	2513.46 ± 302.79	2458.66 ± 350.68	1.001	0.319

Note: DII, dietary inflammatory index

Table 4 Comparison of inflammatory markers between the two groups

Parameters	Low DII group	High DII group	t	*P* Value
C-reactive Protein (mg/L)	3.85 ± 0.86	4.59 ± 1.14	4.339	< 0.001
Interleukin-6 (pg/mL)	5.78 ± 1.22	6.14 ± 1.57	1.537	0.127
Tumor Necrosis Factor-α (pg/mL)	8.91 ± 1.87	9.55 ± 1.14	2.52	0.013
Fibrinogen (g/L)	3.55 ± 0.67	3.89 ± 0.77	2.777	0.006
Erythrocyte Sedimentation Rate (mm/h)	15.69 ± 3.33	16.44 ± 4.69	1.097	0.275
Procalcitonin (ng/mL)	0.54 ± 0.29	0.66 ± 0.39	2.046	0.043
White Blood Cell Count (10^9/L)	6.53 ± 1.58	7.29 ± 1.78	2.701	0.008

Note: DII, dietary inflammatory index

Table 5 Comparison of renal function between the two groups

Parameters	Low DII index group	High DII group	t	*P* Value
Glomerular Filtration Rate (mL/min)	45.68 ± 8.98	42.54 ± 7.87	2.239	0.027
Urinary Albumin Excretion (mg/day)	211.75 ± 21.56	222.69 ± 25.64	2.76	0.007

Note: DII, dietary inflammatory index

calories, sodium intake, and fluid intake between the two groups ($P > 0.05$).

Inflammatory markers

To investigate the inflammatory markers between the two groups, we conducted a study (Table 4). C-reactive protein levels were significantly lower in the low dietary inflammation index group compared to the high dietary inflammation index group with a P value of less than 0.001. Moreover, tumor necrosis factor-α, fibrinogen, procalcitonin, and white blood cell count were also significantly lower in the low dietary inflammation index group compared to the high dietary inflammation index group, with P values of 0.013, 0.006, 0.043, and 0.008, respectively. Meanwhile, interleukin-6 and erythrocyte sedimentation rate did not show statistically significant differences between the two groups ($P > 0.05$).

Renal function

We then compared the renal function between the low dietary inflammation index group and the high dietary inflammation index group, and the results showed that the difference was statistically significant (Table 5). The glomerular filtration rate was significantly higher in the low dietary inflammation index group compared to the high dietary inflammation index group, with a P value of 0.027. Additionally, the urinary albumin excretion was significantly lower in the low dietary inflammation index group compared to the high dietary inflammation index group, with a P value of 0.007.

Correlation analysis

To further explore the relationship between dietary intake and clinical outcomes, we conducted a correlation analysis between protein intake and key inflammatory markers as well as renal function parameters. In the high DII group, protein intake was found to be significantly correlated with CRP levels ($r = 0.572$, $P < 0.001$), indicating a moderate positive correlation between higher protein intake and increased inflammation. Similarly, TNF-α and fibrinogen showed moderate positive correlations with protein intake ($r = 0.460$, i = 0.002; $r = 0.380$, $P = 0.010$, respectively), further supporting the potential pro-inflammatory effects of protein intake in this group.

Additionally, protein intake was positively correlated with procalcitonin ($r = 0.428$, $P = 0.004$) and WBC ($r = 0.310$, $P = 0.030$), indicating a potential relationship between higher protein intake and immune activation markers.

On the other hand, protein intake showed a moderate negative correlation with GFR (r = -0.302, $P = 0.045$), suggesting that higher protein intake may be linked to decreased renal function, especially in the high DII group. Urinary albumin excretion, while positively correlated with protein intake ($r = 0.195$, $P = 0.115$), did not

Table 6 Correlation analysis between protein intake and clinical parameters in the high DII group

Parameters	Correlation Coefficient (*r*)	*P* Value
C-reactive Protein (mg/L)	0.572	< 0.001
Interleukin-6 (pg/mL)	0.315	0.035
Tumor Necrosis Factor-α (pg/mL)	0.460	0.002
Fibrinogen (g/L)	0.380	0.010
Erythrocyte Sedimentation Rate (mm/h)	0.250	0.080
Procalcitonin (ng/mL)	0.428	0.004
White Blood Cell Count (10^9/L)	0.310	0.030
Glomerular Filtration Rate (mL/min)	-0.302	0.045
Urinary Albumin Excretion (mg/day)	0.195	0.115

Table 7 Univariate and multivariate regression analysis results

Parameters	Univariate		Multivariate	
	Coefficient (β)	*P*-value	Coefficient (β)	*P*-value
Glomerular Filtration Rate (mL/min)	-0.191	0.032	-0.182	0.048
Urinary Albumin Excretion (mg/day)	0.231	0.019	0.243	0.014
C-reactive Protein (mg/L)	0.482	< 0.001	0.238	0.023
Interleukin-6 (pg/mL)	0.295	0.044	0.125	0.111
Tumor Necrosis Factor-α (pg/mL)	0.411	0.005	0.109	0.128
Fibrinogen (g/L)	0.289	0.021	0.147	0.082
Erythrocyte Sedimentation Rate (mm/h)	0.180	0.089	-	-
Procalcitonin (ng/mL)	0.355	0.010	0.197	0.037
White Blood Cell Count (10^9/L)	0.271	0.056	-	-
Protein Intake (g/day)	0.352	0.009	0.286	0.018
Fiber Intake (g/day)	0.198	0.062	-	-
Serum Creatinine (mg/dL)	0.623	< 0.001	0.212	0.092
Hemoglobin (g/dL)	0.265	0.042	0.146	0.081
Phosphorus (mg/dL)	0.195	0.080	-	-
Potassium (mEq/L)	0.228	0.065	-	-

reach statistical significance, indicating a weaker association compared to other markers (Table 6).

Univariate and multivariate regression analysis results

Univariate regression analysis showed that DII was significantly associated with several clinical parameters. CRP had the strongest positive correlation with DII (β = 0.482, $P < 0.001$). Additionally, GFR and urinary albumin excretion were significantly associated with DII (r = -0.191, $P = 0.032$ and $r = 0.231$, $P = 0.019$, respectively). Protein intake was also positively correlated with DII ($r = 0.352$, $P = 0.009$), indicating a potential link between higher protein intake and increased inflammatory potential.

In the multivariate regression model, CRP remained significantly associated with DII (β = 0.238, $P = 0.023$). GFR showed a negative relationship with DII (β = -0.182, $P = 0.048$), suggesting that a more pro-inflammatory diet may contribute to reduced kidney function. Urinary albumin excretion and protein intake also remained significant (β = 0.243, $P = 0.014$ and β = 0.286, $P = 0.018$, respectively), reinforcing the association between dietary inflammation and kidney function. Other parameters, such as interleukin-6, TNF-α, and fibrinogen, did not show significant associations in the multivariate model (Table 7).

Adverse events

The comparison of adverse events between the low and high dietary inflammation index groups revealed no statistically significant differences in the occurrence of gastrointestinal symptoms, cardiovascular events, hospitalizations, infections, anemia, bone disorders, and hypotension ($P > 0.05$) (Table 8).

Discussion

The present study aimed to explore the impact of a low DII diet on the clinical parameters of patients with CKD. The findings of this retrospective comparative study shed light on the potential relationship between DII and various clinical parameters, including laboratory markers, renal function, and adverse events in CKD patients. The results suggest that DII may be associated with inflammatory status, laboratory parameters, and renal function in individuals with CKD.

Laboratory parameters such as serum creatinine, hemoglobin, phosphorus, and potassium exhibited

Table 8 Comparison of adverse events between the two groups

Parameters	Low DII group (77)	High DII index group (68)	t	*P* Value
Gastrointestinal Symptoms (%)	8 (10.39%)	9 (13.24%)	0.074	0.785
Cardiovascular Events (%)	6 (7.79%)	7 (10.29%)	0.055	0.814
Hospitalizations (%)	7 (9.09%)	7 (10.29%)	0	1
Infections (%)	4 (5.19%)	5 (7.35%)	0.037	0.847
Anemia (%)	10 (12.99%)	10 (14.71%)	0.003	0.954
Bone Disorders (%)	4 (5.19%)	5 (7.35%)	0.037	0.847
Hypotension (%)	6 (7.79%)	7 (10.29%)	0.055	0.814

Note: DII, dietary inflammatory index

significant differences between the low and high dietary inflammation index groups. Lower levels of serum creatinine and higher levels of hemoglobin in the low dietary inflammation index group may indicate improved renal function and hemoglobin status, respectively. The observed lower phosphorus and potassium levels in the low DII group are noteworthy, as maintaining optimal levels of these electrolytes is crucial in CKD management. These findings suggest that a low DII diet may contribute to the preservation of renal function as well as the management of electrolyte imbalances in CKD patients.

Moreover, the comparison of dietary intake revealed that the low dietary inflammation index group had significantly higher fiber intake and lower protein intake compared to the high dietary inflammation index group. This is consistent with previous research findings [33] that high protein intake may increase glomerular pressure and glomerular hyperfiltration. This may lead to structural damage to the glomeruli, resulting in or exacerbating CKD. Therefore, a low protein diet (LPD) of 0.6–0.8 g/kg/day is typically recommended for the treatment of chronic kidney disease, which can also be used to control metabolic disorders in CKD [34]. However, another study also suggests that high intake of total protein, animal protein, and plant protein is associated with low mortality rates in patients with chronic kidney disease. This suggests that we need to take a more cautious and comprehensive view of protein intake in CKD patients [35]. Moreover, Tomova A et al. showed that higher fiber intake has been associated with anti-inflammatory effects and improved gut microbiota composition, which might contribute to the observed differences in inflammatory markers and renal function between the two groups [36]. On the other hand, the higher protein intake in the high DII group may have implications for uremic toxin accumulation and acid-base balance in CKD, potentially impacting renal function and inflammatory status. These findings highlight the significant variation in protein and fiber intake based on the dietary inflammation index, emphasizing the potential impact of dietary choices on inflammatory markers in patients with CKD.

Inflammatory markers such as C-reactive protein, tumor necrosis factor-α, fibrinogen, procalcitonin, and white blood cell count showed significantly lower levels in the low dietary inflammation index group. These findings of are in line with previous research Garay-Sevilla ME et al. suggesting that dietary components can exert pro-inflammatory or anti-inflammatory effects, thereby influencing the overall inflammatory milieu within the body [16]. The observed differences in inflammatory markers support the concept that dietary choices play a pivotal role in modulating the inflammatory status of individuals and may have implications for the progression of CKD and the development of comorbidities.

Furthermore, the comparison of renal function parameters revealed significantly higher glomerular filtration rate and lower urinary albumin excretion in the low dietary inflammation index group. These findings are of particular significance in the context of CKD management, as preservation of glomerular filtration rate and reduction of urinary albumin excretion are indicative of better renal function and potential slowing of CKD progression. The impact of DII on renal function parameters underscores the potential for dietary to influence disease trajectory and clinical parameters in CKD patients.

The correlation analysis revealed a significant relationship between protein intake and inflammation markers (CRP, TNF-α, fibrinogen, PCT, WBC) in the high DII group, suggesting a potential pro-inflammatory effect of protein intake. Additionally, protein intake was negatively correlated with GFR, indicating a possible link between higher protein intake and decreased renal function in this group. These results are consistent with findings from other studies. For example, a study by Aycart et al. suggests that plant proteins may help reduce inflammation levels, while animal proteins are associated with an increased inflammatory response [37]. In addition, Shankar et al. found that inflammatory markers such as CRP are closely related to the occurrence of CKD [38]. Similarly, Krishnamurthy et al.'s research also supports the link between a high fiber diet and reduced levels of inflammation [39]. However, unlike these studies, the relationship between high protein intake and inflammatory markers as well as renal function decline in this study showed a more direct positive correlation, especially in the high DII group. Overall, these results suggest

that protein intake in the high DII group may exacerbate the course of CKD by promoting inflammatory responses and affecting renal function. This is consistent with previous research, indicating that protein intake in the diet should be given sufficient attention, especially in CKD patients, and controlling protein intake may have the potential to slow down disease progression.

The regression analysis in this study revealed significant associations between DII and CRP, GFR, urinary albumin excretion, and protein intake, with these associations remaining significant in the multivariate model. Although parameters such as IL-6, TNF-α, and fibrinogen did not show significance in the multivariate analysis, these results highlight the important link between dietary inflammation and renal function and inflammatory markers. These findings are consistent with previous research. For instance, a study by Mazidi et al. found a significant association between higher DII and worse kidney function and increased urinary albumin excretion [40]. Similarly, Guo et al. reported a connection between DII and the progression of CKD [41]. However, the negative correlation between protein intake and kidney function, along with the positive correlation with inflammation markers in this study, further emphasizes the impact of dietary patterns on the progression of CKD, particularly the complex relationship between protein intake and renal health. These regression results suggest that DII may play a significant role in influencing inflammatory markers and renal function in CKD patients. While these findings provide valuable insights, further longitudinal studies are needed to validate the causal relationship between DII, renal function, and inflammation.

The comparison of adverse events did not reveal significant differences between the low and high dietary inflammation index groups. While this may appear contrary to the observed differences in laboratory parameters, inflammatory markers, and renal function, it is important to consider the multifactorial nature of adverse events in CKD. Adverse events in CKD are influenced by a myriad of factors, including disease severity, comorbidities, medications, and psychosocial aspects, which may attenuate the specific impact of DII on these outcomes.

The mechanisms by which dietary components influence inflammation and renal function are complex and multifaceted. Diets rich in pro-inflammatory nutrients, such as saturated fats, refined sugars, and processed foods, are known to activate inflammatory pathways through mechanisms like the upregulation of NF-κB and the production of reactive oxygen species (ROS) [42]. These pathways trigger the secretion of pro-inflammatory cytokines, including IL-6 and TNF-α, which play a central role in the pathogenesis of CKD. This inflammatory response is thought to contribute to renal injury by promoting fibrosis, endothelial dysfunction, and a decrease in nephron number, which accelerates the progression of CKD [43]. Conversely, diets high in anti-inflammatory nutrients, such as fiber, polyunsaturated fatty acids, and antioxidants, have been shown to mitigate these inflammatory processes by modulating gut microbiota composition and reducing systemic inflammation [44]. Our findings, which show a significant association between higher DII and increased inflammation markers (CRP, TNF-α), as well as decreased renal function, underscore the impact of dietary choices in CKD patients. These results are in line with studies that highlight the pro-inflammatory effects of a high-protein diet, particularly from animal sources, which can exacerbate kidney damage through the accumulation of uremic toxins and alterations in acid-base balance [38]. The observed negative correlation between protein intake and GFR further suggests that dietary inflammation may contribute to renal function decline, particularly in those consuming higher amounts of inflammatory foods.

Overall, the findings of this study suggest that a low DII diet may be associated with favorable clinical parameters, inflammatory markers, and renal function in patients with CKD. These findings align with the growing body of evidence highlighting the impact of diet on inflammatory pathways and the potential for dietary to mitigate the risk of chronic diseases, including CKD [45, 46]. However, it is important to acknowledge certain limitations of the study, including its retrospective design, relatively small sample size, and potential confounding factors that were not accounted for. Future prospective studies with larger sample sizes and longer follow-up periods are warranted to validate these findings and further elucidate the potential of dietary in optimizing outcomes for CKD patients. Additionally, while the study focused on the DII, other dietary parameters, such as the intake of artificial sweeteners, saturated fats, and carbohydrates, which may also influence inflammation and kidney function, were not evaluated. Further research and clinical trials are essential to establish the efficacy of low DII diet and to inform evidence-based dietary recommendations for CKD patients. Ultimately, the integration of dietary into comprehensive CKD care has the potential to optimize patient outcomes and alleviate the substantial burden of CKD on individuals and healthcare systems worldwide.

Conclusion

In conclusion, the implications of the current study are significant in the context of CKD management and care. The potential relationship between DII and clinical parameters in CKD patients underscores the importance of considering dietary as an integral component of holistic CKD management. Tailored nutritional strategies that focus on modulating inflammatory status through

low DII diets may offer promising avenues for improving renal function, mitigating inflammation, and ultimately enhancing the overall well-being of individuals with CKD. Further prospective studies are warranted to validate these findings and establish evidence-based dietary recommendations for CKD patients.

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Author contributions
All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Weijuan Pan and Jian Feng. The first draft of the manuscript was written by Weijuan Pan and Jian Feng commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was approved by the Ethics Committee of Zhejiang University Affiliated Sir Run Run Shaw Hospital in accordance with regulatory and ethical guidelines pertaining to retrospective research studies. Consent to participate was waived for this retrospective study by the Ethics Committee of Zhejiang University Affiliated Sir Run Run Shaw Hospital due to the exclusive use of de-identified patient data, which posed no potential harm or impact on patient care.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Clinical trial number
Not applicable.

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