RESEARCH ARTICLE

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Does Excessive Meat Consumption During the Feast of Sacrifice Worsen Chronic Kidney Damage? The Effect of Intensive Meat Consumption on Chronic Kidney Damage

ABSTRACT

Objective: Chronic kidney disease (CKD) is precisely described as the availability of kidney damage or decreased kidney fonction that lasts for three months or more, regardless of its cause. Dietary factors may affect the progression and complications of the disease. Our aim is to investigate the effects of excessive meat consumption of Muslims on CKF during the Feast of Sacrifice.

Methods: The study was performed on 203 patients with stage III-V CKD. The biochemical values of the patients who applied to the nephrology outpatient clinic at least one month before and one month after the Eid al-Adha were recorded. Patients who regularly consumed sacrificial meat for 4 days during the feast of sacrifice were included in the study.

Results: The blood urea nitrogen (BUN), creatinine, albumin, potassium (K), calcium (Ca), vitamin D, and base excess (BE) levels of the patients were foundap to be significantly higher after the Feast of Sacrifice ($p=0.014$, $p<0.001$, $p=0.031$, $p<0.001$, $p=0.002$, $p<0.001$and $p=0.009$). The mean e-GFR of the patients before the Feast of Sacrifice was 32.221±14.756. It was found to be 31.474±15.229 after the feast of sacrifice, indicating a statistically significant decrease ($p=0.042$). A slight decrease was observed in the blood pH ($p=0.002$) and bicarbonate (HCO3) ($p=0.002$) levels of the patients. The change in the amount of creatinine with the feast showed a significant difference by CKD stages ($p<0.001$). There was no significant difference between stage IIIa (0.066±0.191), stage IIIb (0.067±0.318) and stage IV (0.137±0.547) patients ($p>0.05$). In stage V patients, creatinine level (0.580±1.124) showed a significant increase ($p<0.001$) due to the short-term intense meat consumption during the Feast of Sacrifice.

Conclusions: In this study, we observed a progressive deterioration in kidney function due to high protein diet caused by excessive meat consumption in a short time during the feast of sacrifice.

Keywords: Chronic Kidney Disease, Feast of Sacrifice, Meat Consumption, Progression of Kidney Disease.

Kurban Bayramında Yoğun Et Tüketimi Kronik Böbrek Hasarını Hızlandırır mı? Yoğun Et Tüketiminin Kronik Böbrek Hastalığına Etkisi

ÖZET

Amaç: Kronik böbrek hastalığı (KBH), nedene bakılmaksızın üç veya daha fazla ay boyunca böbrek hasarı veya azalmış böbrek fonksiyonunun varlığı olarak tanımlanır. Diyet faktörleri böbrek hastalığının ilerlemesi ve komplikasyonları üzerinde etkili olabilir. Çalışmamızda, kurban bayramında toplumda yoğun et tüketiminin KBH progresyonu üzerindeki etkilerini araştırmayı amaçladık.

Gereç ve Yöntem: Çalışma evre III-V KBH tanısı alan 203 hasta üzerinde gerçekleştirildi. Kurban Bayramı'ndan en az bir ay önce ve bir ay sonra nefroloji polikliniğine başvuran hastaların biyokimyasal değerleri kaydedildi. Çalışmaya 4 gün boyunca kurban bayramında düzenli olarak kurban eti tüketen hastalar dahil edildi.

Bulgular: Hastaların kan üre azotu (BUN) ($p=0.014$), kreatinin (Cre) ($p<0.001$), albumin ($p=0.031$) , potasyum (K) ($p<0.001$), kalsiyum (Ca) ($p=0.002$) vitamin-D ($p<0.001$), ve baz açığı (BE) ($p=0.009$) düzeyleri kurban bayramı sonrası anlamlı yüksek bulundu. Hastaların kurban bayramı öncesi e-GFR ortalaması 32.221±14.756 idi. Kurban bayramı sonrası ise istatistiksel olarak anlamlı bir düşüş göstererek 31.474±15.229 oldu ($p=0.042$). Hastaların kan pH ($p=0.002$) ve bikarbonat (HCO3) ($p=0.002$) düzeyinde de bir miktar azalma oldu. Kreatinin miktarında bayram ile meydana gelen değişim ise KBH evrelerine göre anlamlı farklılık gösterdi ($p<0.001$). Evre IIIa (0.066±0.191), evre IIIb (0.067±0.318) ve evre IV (0.137±0.547) hastaları arasında anlamlı fark yoktu ($p>0.05$). Evre V hastalarında ise bayram sürecindeki kısa süreli yoğun et tüketimine bağlı olarak kreatinin düzeyi (0.580±1.124) anlamlı bir artış gösterdi ($p<0.001$).

Sonuç: Bu çalışmada, kurban bayramı boyunca kısa sürede aşırı et tüketiminin neden olduğu yüksek proteinli diyete bağlı olarak böbrek fonksiyonlarında ilerletici bir bozulma olduğunu gözlemledik.

Anahtar Kelimeler: Kronik Böbrek Hastalığı, Kurban Bayramı, Et Tüketimi, Kronik Böbrek Hastalığında Progresyon.

INTRODUCTION

Chronic kidney Disease (CKD) is described as the availability of kidney damage or decreased renal function that lasts for three months or more, regardless of its cause. This disease, which is common worldwide (8% - 16%), is a global health problem that causes millions of deaths each year (1). There are two primary important risk factors for CKD; genetic, environmental. Diabetes mellitus (DM), hypertension (HT), Obesity, infections, inflammation, malnutrition are important causes of environmental factors (2). It is known that food intake is one of the changeable risk factors for kidney dysfunction. Dietary factors may affect the progression and complications of the disease. Among patients with CKD, excessive food consumption causes sodium and volume overload, hypercalcemia, hyperphosphatemia, and accumulation of toxic metabolites generated by protein degradation. On the other hand, poor nutrition increases the risk of malnutrition. However, the most appropriate nutritional approach in patients with CKD is still unknown, and conflicting results have been obtained from the clinical trials conducted so far. The individual optimal diet for patients with CKD varies depending on the estimated glomerular filtration rate (e-GFR), type of CKD (proteinuric or non-proteinuric), and the availability of other comorbidities such as HT either heart failure. Daily protein intake is recommended as 0.8 grams per kilogram of body weight in non-dialysis subjects with CKD (eGFR of <60 mL /min /1.73 m2)(3).

At one study, after 11 years of followup, a western-style diet characterized by processed red meat, saturated fats, and sweets has been shown to worsen kidney function. In the same study, it was claimed that another diet model with a elevated percentage of fruits, vegetables, leguminous plants, fish, poultry, crops preserved kidney function (4).

Appropriate dietary approaches may have a positive effect on clinical outcomes among patients with CKD. However, there is no consensus regarding the most appropriate nutritional approach in patients with CKD, and conflicting results have been reported in the clinical trials conducted so far.

Taking measures get under control the progression and development of complications is of great importance since CKD is an important public health problem. During the Eid al-Adha, there is intense meat consumption in the whole society for 4 days. Our aim is to investigate the effects of excessive meat consumption of Muslims on CKD during the Feast of Sacrifice.

MATERIAL AND METHODS

Before starting the study, approval numbered 2019/128 and dated 12/09/2019 was gained from the Clinical Research Ethics Committee of our university. The study included 203 patients with stage III-V CKD who were admitted to the Nephrology outpatient clinic one month before and one month after the 2020 Feast of Sacrifice (31/07/2020-03/08/2020). All cases consumed 150 grams of red meat (each piece of meat 10 g, a total of 15 pieces 150 grams) at each meal in 3 meals for 4 days during the Feast of Sacrifice . An e-GFR value of less than 60 ml/dk/1.73m^2 was considered as the diagnostic criteria for CKD.The Modification of Diet in Renal Disease (MDRD) study was used to measure e-GFR (5).In accordance with the CKD stages, the patients were classified as stage IIIa (e-GFR of 45-59 ml/dk/1.73m^2), stage IIIb (e-GFR 30-44 ml/dk/1.73m^2), stage IV (e-GFR of 15-29 ml/dk/1.73m^2) and stage V (e-GFR of <15 ml/dk/1.73m^2) (1).At least one month before and after the Feast of Sacrifice, 12-hour fasting blood samples were collected from the patients in the morning. COBAS c501 (Roche, Basel, Switzerland) device was used for routine biochemical analysis (creatinine, C-reactive protein (CRP), uric acid, potassium, sodium, calcium), while CELL-DYN RUBY (Abbott, IL, USA) was used for hemogram test.

Patients with active infections, those who were vegetarian, those who had malignancy, patients with cachexia, and those receiving renal replacement therapy (hemodialysis, peritoneal dialysis) were excepted from the study.

RESULTS

Two hundred three subjects were evaluated in our study. Of all patients, 107 (52.7%) were male and 96 (47.3%) were female. The mean age of the patients was 66.36± 12.91 years, from 19 to 89 years. The mean age of the male and female subjects was 67.81±12.55 and 64.74±13.18 years, respectively (p=0.092).The etiology was diabetes mellitus 81 (39.9%), hypertension 88 (43.3%), and others disease 34 (16.7%). The drugs used by the cases, respectively; 551 Insulin 39 (19.2%), oral antidiabetic 26 (12.8%), angiotensin converting enzyme inhibitors (ACE-I) –angiotensin receptor blockers (ARB) 48 (23.6%), calcium channel blocker 42 (20.7%) and multiple drug use was 48 (23.6%).

The change in the blood parameters before and after the Feast of Sacrifice is presented in Table 1. The blood urea nitrogen (BUN), creatinine, albumin, potassium (K), calcium (Ca), vitamin D, base excess (BE) levels of the subjects were found to be significantly higher after the feast. Following the feast, the BUN level increased from 34.045±15.920 to 35.981±17.633 (p=0.014), the creatinine level increased from 2.378±1.366 to 2.549±1.640 (p<0.001), and the mean albumin level increased from 4.231±0.403 to 4.267±0.406 (p=0.031). The mean e-GFR of the subjects before the feast was 32.221±14.756. It was found to be 31.474±15.229 after the feast, and

this decrease was statistically significant (p=0.042). No statistically significant change was observed following the feast in terms of other blood parameters (p>0.05).

Pearson's correlation coefficient was used to analyze the correlation of creatinine and e-GFR levels with uric acid and albumin levels (Table 2).

Table 1. Changes in blood parameters before and after the Feast of Sacrifice

	n	Before		After		t	p
		Mean	SD	Mean	SD		
Glucose(mg/dl)	203	128.419	57.166	127.665	48.195	0.231	0.818
BUN(mg/dl)	203	34.045	15.920	35.981	17.633	-2.473	0.014*
Creatinine(mg/dl)	203	2.378	1.366	2.549	1.640	-4.065	<0.001
eGFR(ml/dk/1.73m^2)	203	32.221	14.756	31.474	15.229	2.042	0.042*
Cholesterol(mg/dl)	203	191.296	44.636	188.015	45.832	1.462	0.145
HDL(mg/dl)	203	46.064	12.952	45.079	12.123	2.029	0.044*
Triglyceride(mg/dl)	203	181.581	116.310	160.852	90.293	3.323	0.001**
LDL(mg/dl)	197	109.663	36.987	109.351	37.455	0.156	0.876
Albumin(g/dl)	203	4.231	0.403	4.267	0.406	-2.168	0.031*
K(mmol/l)	203	4.853	0.622	5.004	0.629	-3.947	<0.001
Ca(mmol/l)	203	9.172	0.721	9.289	0.687	-3.119	0.002**
P(mmol/l)	203	3.864	0.957	3.891	0.895	-0.578	0.564
CRP(mg/dl)	203	0.918	1.754	0.851	1.330	0.489	0.625
Uric acid(mg/dl)	203	6.609	1.634	6.794	1.659	-1.974	0.051
HB1AC(%)	203	6.283	1.393	6.333	1.440	-1.083	0.280
ACR(mg/g)	202	1153.851	1776.789	1075.581	1731.522	1.097	0.274
BCR(mg/mg)	203	15.754	5.272	15.735	5.325	0.071	0.944
HGB(g/dl)	203	12.049	1.857	12.015	1.934	0.415	0.679
PLT	203	225906.404	77396.025	223157.635	70877.141	1.006#	0.315
Folate(ng/ml)	203	7.960	4.224	7.866	4.488	0.302	0.763
B12 (ng/ml)	203	382.001	260.847	374.630	176.271	0.466	0.642
Ferritin(ng/ml)	203	303.759	447.893	308.059	417.487	-0.295	0.768
PTH(pg/ml)	200	126.945	123.620	124.002	97.756	0.541	0.589
Vitamin D	202	16.034	8.461	17.708	8.520	-3.697	<0.001
PH	203	7.345	0.048	7.335	0.055	3.170	0.002**
HCO3(mEq/L)	203	25.138	3.515	24.667	3.839	2.302	0.022*
PCO2(mmHg)	203	45.855	5.577	46.149	6.331	-0.742	0.459
BE	203	-0.463	4.621	-1.188	4.390	2.642	0.009**

#*Wilcoxon Signed Rank Test Z-Value*, t=Paired t-test

*=<0.05, **=0.01

BUN:The blood urea nitrogen , e-GFR: estimated glomerular filtration rate, HDL: high density lipoprotein, LDL: Low density lipoprotein, K:potassium, Ca: calcium, P: phosphor, crp: C-reactive protein, HB1AC: glycosylated hemoglobin, ACR: Urinary albumin/creatinin ratio, BCR: blood urea nitrogen to serum creatinine ratio , HGB: hemoglobin, PLT: platelet, B12: vitamin B12, PTH: parathyroid hormone,

Table 2. Correlation between subjects' albumin levels and creatinine and eGFR levels

		Correlation Coefficient		Regression Coefficient			
		r	p	b	Std. Error	t	p
Creatinine	Before the Feast	-0.234	0.001**	-0.794	0.232	-3.416	0.001**
	After the Feast	-0.162	0.021*	-0.654	0.281	-2.329	0.021*
e-GFR	Before the Feast	0.323	<0.001	11.815	2.445	4.832	<0.001
	After the Feast	0.284	<0.001	10.632	2.535	4.194	<0.001

e-GFR: estimated glomerular filtration rate

r=Pearson correlation coefficient, b=Linear regression coefficient

*=<0.05, **=0.01

A significant weak negative correlation was detected between creatinine and albumin levels before (r=-0.234, p=0.001) and after the feast (r= -0.162, p=0.021). Likewise, albumin level was found to be correlated with e-GFR. There was a significant weak positive correlation between the albumin levels and e-GFR levels before (r=0.323, p<0.001) and after the feast (r=0.284, p<0.001). In other words, creatinine levels decreased and e-GFR levels increased as the albumin level increased. According to the calculated linear regression coefficients, the creatinine level was estimated to decrease by 0.794 mg/dL (p=0.001) before the Feast and by 0.654 mg/dL (p=0.021) after the Feast

as the albumin level increased by 1 mg. On the other hand, the eGFR level was estimated to decrease by 11.815 mL/min/1.73m^2 ($p<0.01$) before the Feast and 10.632 mL/min/1.73m^2 ($p<0.01$) after the Feast as the albumin level increased by 1 mg. The correlation coefficients calculated between uric acid and both creatinine and e-GFR showed that there was no significant correlation between the variables before and after the Feast ($p>0.05$). The distribution of the subjects before and after the feast by their CKD stages was compared using the McNemar-Bowker test and no significant difference was found ($p=0.091$, Table 3).

One-way ANOVA was carried out to determine at which CKD stage (Stage IIIa, Stage IIIb, Stage IV, Stage V) the amount of change (paired difference) in creatinine,

The blood urea nitrogen (BUN), blood urea nitrogen to serum creatinine ratio (BCR), and eGFR levels after the feast was greater (Table 4).

Table 3. Distribution of subjects before and after the Feast by CKD stages

			After the Feast				Total	p
			Stage IIIa	Stage IIIb	Stage IV	Stage V		
Before the Feast	Stage IIIa	n	38	12	0	0	50	0.091
		%	77.6	20.0	0.0	0.0	24.6	
	Stage IIIb	n	10	45	11	0	66	
		%	20.4	75.0	20.0	0.0	32.5	
	Stage IV	n	1	3	41	8	53	
		%	2.0	5.0	74.5	20.5	26.1	
	Stage V	n	0	0	3	31	34	
		%	0.0	0.0	5.5	79.5	16.7	
	Total	n	49	60	55	39	203	
		%	100.0	100.0	100.0	100.0	100.0	

Stage IIIa: 45-59 mL/min/1.73 m^2; Stage IIIb: 30-44 mL/min/1.73 m^2
Stage IV: 15-29 mL/min/1.73 m^2; Stage V=<15 mL/min/1.73 m^2

Table 4. Descriptive statistics values according to CKD stages

		n	**Mean**	**SD**	**F**	**p**
Creatinine	Stage IIIa	50	0.066[b]	0.191	7.099	<0.001
	Stage IIIb	66	0.067[b]	0.318		
	Stage IV	53	0.137[b]	0.547		
	Stage V	34	0.580[a]	1.124		
BUN	Stage IIIa	50	1.666	6.189	0.820	0.484
	Stage IIIb	66	0.421	7.422		
	Stage IV	53	3.077	16.013		
	Stage V	34	3.497	13.544		
BCR	Stage IIIa	50	.261	3.869	0.821	0.493
	Stage IIIb	66	-.534	3.476		
	Stage IV	53	.475	4.584		
	Stage V	34	-.200	2.760		
eGFR	Stage IIIa	50	-2.004	5.935	1.556	0.201
	Stage IIIb	66	-0.116	6.181		
	Stage IV	53	-0.168	4.360		
	Stage V	34	-1.024	2.206		

BUN:The blood urea nitrogen , BCR: blood urea nitrogen to serum creatinine ratio, e-GFR: estimated glomerular filtration rate, Stage IIIa: 45-59 mL/min/1.73 m^2; Stage IIIb: 30-44 mL/min/1.73 m^2
Stage IV: 15-29 mL/min/1.73 m^2 ; Stage V=<15 mL/min/1.73 m^2
F= One-way ANOVA
Means that do not share a common letter are significantly different ($p<0.001$).

There was no significant difference between the CKD stages in terms of the change in e-GFR, BUN, and BCR levels after the feast ($p>0.05$). The change in the amount of creatinine with the feast showed a significant difference by CKD stages ($p<0.001$). There was no significant difference between subjects with stage IIIa (0.066±0.191), stage IIIb (0.067±0.318), stage IV CKD (0.137±0.547) ($p>0.05$), while a significant increase was detected in creatinine levels (0.580±1.124) of subjects with stage V CKD due to the short-term excessive meat consumption during the feast ($p<0.001$).

DISCUSSION

The current study demonstrated that extraordinary excessive meat consumption for a short time during the Feast of Sacrifice worsened the kidney function in subjects with stage III-V CKD.

Chronic Kidney Disease is a majority disease affecting approximately 10–13% of adults (6). It is thought to cause a partial increase in traditional risk factors including HT, DM, and metabolic syndrome, and to significantly increase cardiovascular risk. The protein intake has been one of the most discussed dietary practices in CKD. Urea, creatinine, and uric acid, degradation products caused by protein metabolism are eliminated by the kidneys. Kidneys are involved in amino acid metabolism through the conversion of phenylalanine to tyrosine and of glycine to serine as well as in the extraction. Therefore, certain abnormalities occur in amino acid plasma concentrations in CKD. There is not enough common consensus about the restriction of certain foods or supplementation of certain nutrients affecting the morbidity or mortality of kidney failure. (7).

A healthy diet is related to lower mortality rates in subjects with CKD. Protein amount of 0.8 g/kg/day, which does not cause malnutrition or progression, is advised for non-dialysis CKD subjects with e-GFR of less than 60 mL/min/1.73m^2 (3). Increasing the consumption of fruits, vegetables, fish, and legumes and cutting down the amount of red meat, refined sugar, and sodium intake can be effective to lower mortality rates in people with renal failure. A balance between protein metabolism and protein intolerance or protein poisoning should be established in subjects with CKD. Limiting protein intake in diet has been a common practice used to control uremia and delay the progression of CKD (6). A meta-analysis suggested that low dietary protein intake decelerate the progression of CKD and residual renal function loss in subjects with advanced stage CKD (7). In a study by Fouque et al. (8) investigating a high-protein diet in subjects with CKD, the mortality rate in subjects with no protein restriction in their diet was found to be approximately 31% compared to those treated with a low protein diet. Kasiske et al. (9), asserted dietary low protein intake delayed the decline in kidney function, however was not sufficient alone.

Krebs et al. (10) reported that a low-carb and highly protein nourishment had no negative act upon creatinine and e-GFR levels. Larsen et al. (11) observed that a high observed that a high-protein diet caused no increase in creatinine levels in type 2 DM subjects with normal kidney functions.In a study by Tirosh et al. (12), an increase was reported in e-GFR levels of subjects who were following a high-protein diet, particularly in those with stage III CKD.In a study by Knight et al. (13), no high-protein diet-induced change was observed in e-GFR levels in cases with normal kidney function and high intake of non-dairy animal protein was related to increased e-GFR reduction in mild CKD.Cirillo et al. (14) described that high protein intake was cross-sectionally associated with higher GFR in middle-aged adults but was related to even greater GFR decline in the long term. Lew et al. (15) suggested that red meat consumption elevated the hazardous of end-stage renal disease.Similarly, unusual high red meat intake, albeit for a short time, has been observed to increase the loss of kidney function (increase in BUN and creatinine levels; decrease in e-GFR) in the present study. The progression in creatinine level has been observed to be more pronounced in non-dialysis subjects with stage V CKD.

Kidneys have a significant role in maintaining potassium homeostasis. The risk of hyperkalemia is higher in subjects with CKD, which is an important cause of comorbidities, such as cardiac arrhythmia and arrest, and mortality. The average potassium intake is 50-100 mEq/day in the Western pattern diet .The factors that determine the plasma potassium concentration are the amount of potassium intake in the diet, potassium distribution between cells and extracellular fluid, and the relation among plasma potassium and urinary potassium excretion.In the literature, the prevalence of hyperkalemia in CKD subjects has been reported to range from 1% to 50%, depending on the threshold level of serum potassium used for diagnosis (16). Diets rich in plant-based foods such as vegan or vegetarian diets have positive effects in subjects with CKD compared to diets high in alkalis and fiber (19).An elevated in serum potassium levels of subjects with CKD is a uncommon symptom in subjects following a vegetarian diet with low protein content (17). In line with the literature data, a high protein diet was observed to cause hyperkalemia in the present study. Although limiting potassium-rich foods is very important in subjects with CKD, attention is uncommonly paid to this during clinician visits. There is a need for studies investigating the effect of potassium diet management in subjects with severe CKD.

Chronic Kidney Disease is described as a e-GFR level less than 60 mL/min/1.73 m^2 or an urine albumin/creatinine ratio greater than 30 mg/g (5).Although there are studies observing no change in the urine microalbumin level despite high-protein diet (11,12), Friedman et al. (18) reported an increase in proteinuria.In the present study, no change was observed in the amount of urine ACR in subjects with CKD, who followed a high-protein diet. Our study is consistent with the literature in this regard.

Maintaining pH balance is one of the kidney functions. Decreased GFR in subjects with CKD is the foremost risk factor for metabolic acidosis. Furthermore, each 1 g/dl increase in albumin is the cause of a 35% increase in metabolic acidosis (19) .A fruit and vegetable-based diet that reduces kidney acid load by 50% can reduce kidney damage, including albuminuria (20). Metabolic acidosis is associated with increased risk of adverse kidney outcomes and mortality in patients with

non-dialysis dependent chronic kidney disease (21).In this study, a high protein diet was detected to worsen metabolic acidosis in subjects with CKD. Our study is consistent with the literature in this regard.

High FGF-23 levels inhibit 1α-hydroxylase activity as kidney functions decrease, and the resulting gastrointestinal absorption of decreased 1.25(OH) vitamin D_2 and calcium results in hypocalcemia (22). In the present study, the high protein diet was observed to have a positive effect on vitamin D and calcium. Considering the harmful effects of a high-protein diet, attention ought to be paid to this, the long-term results of which are unknown. In our study, although we found low triglyceride levels, no positive change was observed in LDL and total cholesterol levels. Studies with larger populations and longer follow-up are needed for the effects of high consumption of red meat on lipid metabolism during Feast of Sacrifice.

This study has several limitations. Firstly, the amount of red meat consumed by the subjects was not clear and the study was conducted in a regional area. Secondly, it was the limitation of our study that it was not taken into account what kind of meat consumption (cow, sheep) was. Another limitation of our study was that CKD etiologies were not questioned. Finally repeat measurements were not performed to determine the long-term effects of sacrificial meat consumption on kidney function.

CONCLUSION

In conclusion, subjects with CKD had a progressive deterioration in their kidney functions due to the high-protein diet caused by excessive meat consumption during the Feast of Sacrifice.A marked deterioration was observed in the blood gas besides the renal dysfunction. Considering the progression of CKD, the positive effect seen in vitamin D and calcium levels is negligible. The result that we found in our study, that acute intense meat consumption adversely affects renal functions, reminds us to be very careful about this issue. We believe that a protein-restricted diet not causing protein malnutrition in subjects with CKD may have positive effects on the course of the disease, and that clinicians need to follow their subjects more carefully. There is a need for studies with a larger patient population. We believe that our study will shed light on future studies to be conducted with larger populations.

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