The Nutritional and Biochemical Indicators of Cardiovascular and Chronic Kidney Diseases Patients Compared to a Normal Group

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Open Access

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Received: 20 April 2024
Accepted:23 May 2024
Published online: 14 June 2024

Citation
Mohamed MA; Emara AH; Ibrahim NR; Ahmad EM; Abd El-Hamid and El-Sayed HH (2024): The Nutritional and Biochemical Indicators of Cardiovascular and Chronic Kidney Diseases Patients Compared to a Normal Group,BNNI (63) 51 - 76.doi. 10.21608/BNNI.2024.360774

Chronic kidney disease (CKD) and cardiovascular disease (CVD) exacerbate each other's progression, increasing morbidity and mortality rates. Studying these interrelated conditions requires strict nutritional and biochemical monitoring. By comparing macro- and micronutrient intake to the recommended daily intake (RDI), biochemical tracking provides valuable information about metabolic diseases, aiding early diagnosis and tailored treatment regimens. This study involved 60 subjects: 30 healthy controls and 30 patients with CKD and CVD from the outpatient clinic of the National Institute of Urology and Nephrology in Cairo, Egypt. The findings demonstrated that macronutrient intake in both groups was lower than the RDI except for protein. Both groups had inadequate vitamin D (VD) taken, getting smaller than 50% of the RDI, with significant differences noticed in serum VD levels, which were less in the CKD+CVD group. Except for sodium intake, higher than the RDI in both groups, all mineral intakes were below the RDI, with magnesium and potassium intake less than 50% of the RDI. Biochemical markers of renal mission, except for the estimated Glomerular Filtration Rate (eGFR), were lower than normal in the patient group. The patient group had greater concentrations of cardiac markers such as creatine kinase, troponin, lactate dehydrogenase (LDH), and vascular cellular adhesion molecule-1 (VCAM-1). Their lipid profile showed significant increases in triglycerides, low-density lipoprotein (LDL-C), very LDL-C, atherogenic index (AI), and atherogenic coefficient (AC), while high-density lipoprotein (HDL-C) levels significantly decreased. This study emphasizes that nutritional intervention and novel biomarkers are crucial for these patients.

Keywords: *Chronic Kidney and Cardiovascular Diseases patient – Nutrition –Biomarker*

INTRODUCTION

The linked health disorders CKD and CVD provide major obstacles to the world's medical infrastructure. The coexistence of CKD and CVD exacerbates morbidity and mortality rates, necessitating vigilant surveillance strategies to manage patient outcomes effectively. Recent epidemiological data highlight the widespread prevalence and impact of these conditions, emphasizing the urgency for comprehensive care approaches. For instance, according to the study conducted by **Mills et al (2015),** Roughly 9.1% of people worldwide suffer from CKD, which greatly increases the incidence of chronic diseases globally. Simultaneously, CVD remains the leading cause of mortality globally, as evidenced by reports from the **WHO in 2021; and GBD in 2020**. These statistics underscore the critical need for targeted interventions and personalized care strategies in managing patients suffering from both CKD and CVD.

Nutritional and biochemical surveillance plays a crucial role in managing patients with CKD and CVD. Adequate nutritional status is critical, as malnutrition and imbalanced diets can exacerbate both kidney and cardiovascular dysfunction **(Ikizler et al., 2021).** Specifically, dietary focusing on sodium restriction, and protein intake, are critical for optimizing patient outcomes **(Kim and Jung 2020); (Kalantar-Zadeh and Fouque 2017)**

Studies by **Kim and Jung (2020) and Mozaffarian and Others (2012)** underscore the focal role of dietary factors in modulating the risk and progression of both CKD and CVD, highlighting the need for tailored nutritional strategies. Monitoring parameters such as protein intake, sodium restriction, and micronutrient status are essential in optimizing metabolic balance and cardiovascular health in this vulnerable population.

Biochemical observation is essential for the early detection of metabolic abnormalities and guiding therapeutic interventions in patients with CKD and CVD. Recent advancements in biomarker research have provided insights into the pathophysiological mechanisms underlying these diseases, enhancing physicians' ability to monitor disease progression and treatment response. **Stopic et al. (2022)** have identified novel biomarkers and biochemical pathways implicated in CKD and CVD, offering potential targets for prognostication and personalized management. Biomarkers that serum

creatinine, eGFR, lipid profiles, and inflammatory markers serve as valuable tools for risk stratification and therapeutic decision-making in this patient population. Integration of advanced biochemical assays and multi-omics approaches holds promise in refining risk assessment and tailoring precision medicine approaches for patients suffering from both CKD and CVD. Continued research and innovation in this field are essential to develop more effective monitoring techniques and therapeutic interventions, ultimately aiming to reduce morbidity.

STUDY AIMED

The research problem sought to address the following questions within the scope:

- How do the nutritional profiles of patients with CVD and CKD differ from those of individuals without these conditions?
- What are the biochemical indicators that exhibit significant variations between patients with CVD and CKD, and individuals with normal conditions?
- Are there specific biomarkers or biochemical parameters that serve as reliable indicators of the nutritional status in patients with CVD and CKD?

SUBJECTS AND METHODS:

For this study, sixty volunteers between the ages of 45 and 60 were selected from the National Institute of Urology and Nephrology's Department's outpatient clinic in Cairo, Egypt. Two categories were created from the cases: Group 1 (n=30) consisted of healthy individuals, a typical group. Group 2 (n = 30) patients with CVD and CKD. Age and sex are matched through two groups.

Inclusion Criteria

KDIGO (2012) guidelines should be followed for the screening and management of patients with CKD. The following was done using an eGFR to categorize CKD patients into stages 5: Stage 1: GFR > 90 mL/min, either normal or high. there are four phases of CKD: mild in the second phase

(GFR = 60–89 mL/min), moderate in stage three (GFR = 30-59 mL/min), severe in the fourth stage (GFR = 15–29 mL/min), and end-stage in stage five (GFR~ 15 mL/min). These are the same people who have been diagnosed with CVD based on electrocardiography (ECG) and blood cardiac enzyme levels, including troponin and CK-MB.

Exclusion Criteria:

This work excluded participants with malignancies, muscular problems, pancreatitis, diabetes mellitus, alcoholism, and smoking.

Dietary Evaluation:

The study participants were evaluated using a twenty-four-hour food recall. The energy and macro/micronutrient content of the 24-hour meal intake were determined using the food configuration tables published by **NNI, (2006)**. The required daily intake (RDI) was compared to all nutrient content of food and beverages consumed, based on the conclusions of the joint Food and Drug Administration (FDA) expert opinion on human nutrient requirements (**FDA, 2020).**

Laboratory tests:

a) The equation declared by **Levey and Else (2000)** was used to calculate serum eGFR as follows:

Estimated GFR = 141 x min (S_{Cr}/κ, 1)$^{\alpha}$ x max (S_{Cr}/κ, 1)$^{-1.209}$ x 0.993^{Age} x 1.018 [if female] x 1.159 [if Black] SCr (standardized serum creatinine) = mg/dL., K = 0.7 (females) or 0.9 (males), α = −1.329 (female) or−0.411 (male), Min = indicates the minimum of S_{Cr}/K or 1, max = indicates the maximum of S_{Cr}/K or 1, Age = Years

b) The colorimetric method of **Trinder (1969)** was applied to gauge serum total cholesterol; **Bucolo and David (1973)** used the same colorimetric technique to criterion serum triglycerides; **Trinder (1969)** used the same colorimetric analysis to estimate serum HDL-C level; **de Cordova and de Cordova (2013)** used the formula to calculate serum LDL-C as follows:

LDL- c (mg/dl) = TC- HDL-c –(TG/5)

c) Mougios, (2007) states that the Auto analyzer (BT1500) was utilized to estimate the creatinine kinase MB.

d) According to **Giannitsis et al. (2010),** the Cardiac Troponin was measured by a Cobas e411 analyzer (Roche, Germany). By **Tietz et al., (1983),** the auto-analyzer (BT1500) was used to estimate the cardiac lactate dehydrogenase.

e) ELISA was used to detect the serum levels of vascular adhesion molecule-1 (VCAM-1) **(Nagel et al., 1994),** vitamin DBP **(Chun, 2012),** and VD (1,25-Dihydroxy VD) **(Holick et al., 2024).**

f) The current equation was calculated as the atherogenic index (AI):

AI=[(log 10 [TG)/HDL-c] according to **Nosrati et al. (2021**). Another metric is the atherogenic coefficient (AC), which is deliberate by the equation

AC = (TC−HDL-c) / HDL-c by https://calculator.academy/atherogenic-coefficient-calculator.

Analyzing Statistical Data:

The data was evaluated using the Statistical Package for the Social Sciences (SPSS, version 22). The descriptive mean ± standard error (SE) or standard deviation (SD) findings were obtained. A t-test paired two samples for means was used to analyze the data. According to **Chan (2003)**, a difference was judged statistically big when *P* was lower than 0.05.

Ethical considerations

The Scientific Research Morals Board confirmed the research of the General Authority for Hospitals and Educational Institutes under the reference number (IUN 00023).

FINDINGS AND DISCUSSION

Table (1) displays the distribution of the sample's criteria, CKD proteinuria stages, and dialysis between the two patient sets. In the normal group, there were more females and fewer meals than in the patient group. The patient group consisted of obese people (BMI = 30.8). In the patient group, stage 5 CKD had the largest percentage (46.7%). where 10% of group 2's urine contains traces of protein and 36.7% of them have positive proteinuria. On the other hand, 56.7% of G2 patients underwent dialysis.

Protein amount was consumed more than what was advised, while all macronutrient intake was below the RDI **(Table 2).** Group (2) (CKD and CVD) patients ingested more calories than the normal group. Patients in group 2 ate large amounts of protein. However, consumption of fat and fiber remained unchanged. It is advised to consume 25–35 kcal/kg of calories per day to maintain a neutral or positive nitrogen balance and to offset the increased energy expenditure that occurs during rest because of comorbidities and inflammation (**Chan et al., 2017).**

Remarkably, patients in group 2 consumed more calories than the normal group. The conventional wisdom that patients should maintain and enhance their nutritional status while unwell may contribute to this in part. Some have hypothesized that patients who are overweight or obese may receive better and more frequent care in addition to leading healthier lives than patients with other conditions (**Wang et al., 2019).** However, consuming large amounts of carbs may also be the cause of this elevated calorie value. It greatly improved by cutting back on the amount of carbohydrates consumed after being conscious of nutrition. Since extra calories are retained as fat,

high-calorie diets are at all events harmful to the renal (**Rios et al., 2018).** This is because high-calorie diets increase fat or carbohydrate intake. The accumulation of fat cells and their increased adiposity in tissues are the causes of obesity and are harmful to health **(Braun, 2017).**

The findings in **Table (3)** demonstrated a significant amount of vitamin D while C consumption was not significant between the 2 groups. Contradictory findings have been found in earlier research on the relationship between vitamin C intake and renal illness **(Ferraro and Another, 2016; Farhadnejad and Colleagues 2016).** The development of CKD stage three or above was linked to the increase in vitamin C consumption when compared to the recommended range. The current findings were indirectly supported by a previous study that linked excess vitamin C taking above the recommended threshold (90 mg/day) to a higher risk of renal gravels (**Ferraro and Others 2016). Lamarche et al., (2011)** reported that despite conflicting epidemiological data, a biologically plausible association exists between increased vitamin C intake and decreased eGFR. Patients with CKD should not take more than 100 mg of vitamin C daily due to the possibility that metabolic by-products of vitamin C could build up and impair renal function**.** reported that vitamin C's metabolites accumulate and impede the activity of the kidneys, patients with CKD shouldn't consume over one hundred mg/day of the vitamin.

In both groups, VD assimilation was decreased by 50% of RDI **(Table 3)**. **Kaur et al. (2019)**, said low VD consumption leads to endothelial dysfunction and inflammation. **Gluba-Brzózka and Partner, (2018)** found a high link between vitamin D therapy and improved outcomes in those on hemodialysis. Even in the initial phases of chronic renal disease, there is a deficit of vitamin D. According to experimental research low levels of VD have been linked to cardiac contractile failure, boosted heart mass, and raised myocardial collagen content.

Table (4) presents statistical data that demonstrates a major ($P<0.05$) variation in the phosphorus intake of each group. This table showed that the intake of iron and zinc was more than 50% from RDI and did not differ statistically between the two groups, as did the tables for other minerals. Intake of magnesium and potassium was less than 50% of RDI in

both groups. In contrast, sodium intake was higher in the sick and normal groups, respectively, than in the RDI (122.8 & 155.2%). In both groups, calcium intake was nearly fifty percent of RDI.

Due to decreased iron intake, increased metal losses, and poorer mobilization of iron from reserves, patients with CKD, especially those on dialysis, show substantial alterations in iron balance and tissue division **(Wish and Teammate 2018)**. Iron homeostasis failure is a major cause of anemia in individuals with CKD if untreated. According to **Kristensen et al., (2019),** zinc is an essential mineral that controls the expression of several genes, including those related to antioxidant resistance, microglial immunity, and antiviral and antibacterial immunity.

A crucial mineral with catalytic, metabolic, and categorical functions is zinc. Deoxyribonucleic acid (DNA) replication, cell proliferation, energy consumption, and growth are among the enzyme-dependent processes in which zinc is engaged **(Macdonald 2000; Haase and Rink 2014).** Additionally, proteins' stability and structure depend on zinc **(Laitaoja and Coworkers 2013).** Maintaining the structure and makeup of cell membranes is also necessary (**Maret, 2017).** Recent research indicates that zinc also has a role in the regulation of leptin, insulin signaling, and antioxidant activity **(Cheng and Chen, 2021; El-Adl et al., 2024)**.

Dietary phosphorus restriction is recommended as a technique for treating hyperphosphatemia because it has been associated with renal failure in individuals with CKD (**Bellasi et al., 2011; Chang and Anderson 2017).**

An increased dietary K+ consumption is a therapeutic challenge for patients with hyperactive kalemia-affected late-stage CKD **(Palmer and Clegg 2016; Kovesdy et al., 2014).** Dietary K+ restriction (3 g/d) is recommended for people who are at risk of hyperkalemia; however, it should only be used individually because it can prevent patients from benefiting from heart-healthy diets (**Rastogi and Colleague workers 2016; Cupisti and another, 2018; Kinsella and Associate, 2010)**. However, it has been suggested that a high-potassium diet may be preventive against CKD because of its physiological effects, which include lowering blood pressure (BP) or acting as a vascular protectant **(Wang et al., 2007).** Patients with severe CKD should take up to 4700 mg of potassium per day, according to prior

research **(Aaron and Sanders, 2013; Kim and Colleagues 2018).**

One modifiable factor that can affect a patient's risk of both cardiovascular disease and the course of their CKD is their dietary sodium consumption. The impact of salt consumption on fluid overload and hypertension—two factors that are predictive of renal advancement and cardiovascular remodeling—has been shown in earlier studies. Furthermore, a high salt diet may harm blood arteries **(Suckling et al., 2010; Gradual and Colleagues 2017; McMahon et al., 2013).**

Notably significant (P<0.001) differences were observed in the studied biochemical parameters for assessing renal functions and eGFR between CKD patients with complicated CVD and the normal group (**Table 5).** Kidney and cardiovascular health are negatively impacted when uremic solutes are retained due to reduced kidney excretory function **(Velasquez et al., 2018).** The epithelial bonds that keep together luminal cells are weakened when gut bacteria convert urea to ammonia. According to **Vaziri et al. (2013),** this makes it possible for germs to enter the bloodstream and cause systemic inflammation, which may accelerate the onset of chronic kidney disease (CKD).

Contrasting group 2's cardiac parameters to those of the healthy category (G1), **Table (6)** shows a substantial (P<0.05) rise. even though VCAM-1 and LDH were within normal limits. Significant increases in VCAM levels (P<0.001) were observed in G2. In the patient population, elevated levels of cardiac activity-related markers (CK-MB, LDH, and troponin) signify myocardial damage. Increases in these values in serum point to modifications in the heart membrane's integrity and permeability. The increase in the proinflammatory VCAM-1 indicates that these changes in the membrane could be brought on by the inflammatory state. Neutrophils can move and translocate across the intercellular link due to modifications in the cytoskeleton of the cellular endothelium and increased vascular permeability **(Van Wetering et al., 2003).** The endothelium's integrity may be further jeopardized by the interaction between neutrophils and VCAM-1, which is considered to activate inflammatory cells and induce transcellular permeability pathways that change the glycocalyx of the endothelium by releasing proteinases.

Data shown in **Table (7)** show that the patient group (G2) had significantly higher blood levels of triglycerides, LDLC, VLDL-C, AI, and AC. Furthermore, when compared to the normal group, the HDL-C levels in (G2) were significantly lower. There were appreciable increases in the serum levels of LDL-C and AC value between the CKD + CVD group and the normal individuals. Serum cholesterol levels between the two groups showed significant differences as well, while they were still within the normal range.

One of the most prevalent side effects of a chronic kidney disorder is dyslipidemia, which typically occurs concurrently with declining kidney performance. It advances according to the CKD stage **(Saini et al., 2022). Mikolasevic and Fellow workers (2017)** indicated that hypertriglyceridemia, primarily caused by elevated lipoprotein triglyceride levels and prolonged triglyceride breakdown, is the most common lipid anomaly in patients with CKD**. Hirano and Collaborators (2003)** displayed that triglyceride levels are higher in individuals with CKD due to decreased breakdown of triglyceride-rich lipoproteins caused by elevated serum levels of a protein known as C (Apo C). Cardiovascular events have been linked to elevated triglycerides and decreased HDL-C in patients with CKD **(Turak et al., 2016).** In a similar vein, high levels of triglyceride-rich lipoproteins are associated with lipid profiles in individuals in the later stages of CKD **(Lamprea-Montealegre et al., 2018).** While LDL-c and atherosclerotic events correlate in the general population, in patients with end-stage renal disease (ESRD), LDL-c has a negative correlation and a flat or weakly positive correlation with mortality at levels below the average and above the average. Those with CKD and kidney transplant recipients benefit from lowering LDL-C, whereas dialysis patients do not **(Ferro and Workmate 2019).**

Latest scientific research, according to **Zewinger et al. (2017),** suggests that the vascular effects of HDL-c can vary depending on the condition and that the quality and composition of blood lipids, particularly triglyceride-rich lipoproteins, and HDL-c, are significantly altered as kidney damage progresses, favoring an atherogenic profile. Adverse endothelial effects from HDL-c may also be observed in children with CKD who do not yet have cardiovascular risk factors such as diabetes, smoking, hypertension, or

dyslipidemia **(Shroff, and Others 2014); (Jankowski and Else 2021).**

Table (8) data showed a significant decrease in group 2's serum vitamin D levels. VDBP was non-significant in the two groups. CKD impairs the kidneys' capacity to carry out specific endocrine functions appropriately. Vitamin D metabolism is known to be mostly regulated by the kidneys, which convert vitamin D into its active form, 1,25-dihydroxy-VD, or calcitriol. Among many other reasons, dietary restrictions and reduced sun exposure increase the risk of multifactorial VD insufficiency in individuals with CKD. It is now widely acknowledged that VD deficiency affects many people globally **(González-Parra and Colleagues 2012; Jean et al., 2017).**

A significant proportion of people with chronic kidney disease (CKD), accounting for about 80% of pre-dialysis patients, suffer from vitamin D insufficiency. This deficit usually starts early in the illness and worsens when the kidneys fail (**Caravaca-Fontan et al., 2016; Cardoso and Pereira, 2019).** The Kidney Disease Improving Global Outcomes (KDIGO) group recommends Vitamin D supplementation. However, the agents or best practices for regaining vitamin D levels are not specified. In individuals with CKD, this impairment is caused by multiple variables that impact its metabolism's synthesis, activation, and degradation stages. The strategy for vitamin D supplementation in CKD has changed as clinical knowledge of its significance has grown to guarantee sufficient availability. With advancing CKD stages and declining renal mass, the capacity to produce active 1,25 vitamin D decreases, leading to its deficiency. To address this, supplementation with calcitriol or its analogs becomes necessary to compensate for compromised production in later CKD stages (beyond Stage 3), enabling the fulfillment of classical hormonal functions **(Williams et al., 2009).**

In the current study, anomalies in Vitamin D Binding Protein (DBP) and renal vitamin D metabolism were noted in CKD patients. According to this study, there is a correlation between decreased phosphorus excretion and active vitamin D production and the increasing reduction in renal function. Additionally, poor vitamin D status in uremic patients can stem from factors such as decreased appetite, gastrointestinal issues, limited dietary intake due to restrictions (like low-protein and low-phosphate diets), and reduced sun exposure due to mobility limitations **(Yoon et al., 2019).** CKD

patients exhibit a diminished response to high-dose cholecalciferol supplementation due to impaired renal vitamin D hydroxylation, exacerbating the deficiency. Research by **Del Valle et al., (2007)** found that 84% of hemodialysis (HD) patients with vitamin D insufficiency lacked sufficient sun exposure.

CONCLUSION:

This study's monitoring of dietary intake revealed that low nutrient intakes, such as those of fiber, potassium, magnesium, vitamin D, and C, as well as many high nutrient intakes such as those of protein, calories, fat, salt, and phosphorus, harmed the patient's CVD and CKD. For these patients, nutrition intervention is therefore essential. Novel biomarkers and metabolic pathways connected to CKD and CVD are potential targets for prognostication and customized treatment. The ultimate goal of lowering morbidity necessitates further investigation and creativity in this field to provide more effective therapy strategies and monitoring techniques.

RECOMMENDATIONS:

- Individualized Nutritional Interventions:

Develop personalized nutrition plans for patients with CKD and CVD, ensuring a balanced intake of essential nutrients to mitigate negative impacts associated with their excesses or deficiencies.

- Regular Monitoring:

Implement routine biochemical investigations to monitor key indicators for ills. This will help in early detection and timely management of disease progression.

- Education and Support:

Provide comprehensive education and support programs for patients and caregivers about the importance of nutrition in managing CKD and CVD. This should include practical advice on dietary modifications and the potential impact of different nutrients on health.

- Research and Innovation:

Continue research to identify and validate novel biomarkers and biochemical pathways associated with CKD and CVD. This will enhance prognostication and allow for more tailored treatment approaches.

Promote innovation in therapeutic strategies and surveillance methods to improve the efficacy of interventions and reduce morbidity rates.

- Interdisciplinary Collaboration:

Foster collaboration between nephrologists, cardiologists, dietitians, and other healthcare professionals to

ensure a comprehensive and integrated approach to patient care.

By implementing these recommendations, healthcare providers can enhance the quality of care for patients suffering from CKD and CVD, ultimately improving patient outcomes and quality of life.

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Table 1. Distribution of criteria of the sample, stages of CKD, proteinuria, and dialysis in the studied groups

Parameters		G1 Normal group		G2 Patient group	
		Count	%	Count	%
Sex	♂	10	33.3	17	56.7
	♀	20	66.7	13	43.3
Age	range	45-60		45-60	
BMI	Mean	24.8		30.8	
CKD stages	2	0	00	3	10
	3	0	00	4	13.3
	4	0	00	9	30
	5	0	00	14	46.7
Proteinuria	+ve	0	0.0	11	36.7
	trace	0	0.0	3	10
	Nil	30	100	2	6.6
Dialysis	Yes	0	0.0	17	56.7
	No	30	100	13	43.3

BMI is calculated using the equation kg/m2, where kg is an individual's weight in kilograms and m2 is the square of their height in meters. Underweight: BMI <18.5; Normal weight: BMI >18.5–24.9; Overweight: BMI =25–29.9; Obesity: BMI ≥ 30

Table 2. Distribution of both macronutrient intake /day (mean ± SE) and % from RDI calculated according to these values means in studied groups.

Macronutrients	G1 normal group	G2 patient group	*P*-value
Calories/kcal	1446.7±97.3	1773.0±102.9*	0.000
RDI	**2000 kcal**		
% from RDI	72.3%	88.7%	
Protein /g	70.2±5.2	72.6 ±6.5	0.38
RDI	**50 g**		
% from RDI	140%	145%	
Fat /g	59.5±5.6	60.7±5.8	0.33
RDI	**78 g**		
% from RDI	76.3%	77.8%	
Carb. /g	163.5±14.5	233.4±12.9*	0.002
RDI	**275 g**		
% from RDI	59.5%	84.9	
Fiber	8.9±1.5	9.2±1.2	0.77
RDI	**28 g**		
% from RDI	31.8%	32.9%	

**At P < 0.05, the mean difference is significant. RDI as stated by the FDA in 2020*

75–100% of the RDIs are considered adequate consumption levels, while fewer than 50% of the RDIs are deemed insufficient intake levels.

Table 3. Distribution of Vitamins C and D intake /day (mean ± SE) and % from RDI calculated according to these values means in different studied groups.

Vitamins	G1 normal group	G2 patient group	P value
Vitamin C mg/day	28.9±8.0	36.8±11.4	0.296
RDI	**90 mg/day**		
% from RDI	32.1%	40.9%	
Vitamin D IU/day	56.5±7.1	34.9±2.9*	0.004
RDI	**800 IU/day**		
% from RDI	7.1%	4.4%	

**The mean difference is Significant at P < 0.05. RDI according to FDA 2020 Intake levels of 75- 100 percent of the RDIs are regarded as adequate, whereas levels of less than 50 percent of the RDIs are deemed insufficient.*

Table 4. Distribution of minerals intake /day (mean ± SE) and percent from RDI calculated according to these values means in different studied groups.

Minerals	G1 Normal group	G2 Patients' group	P value
IRON (mg /day)	11.4±0.8	13.2±0.7	0.07
RDI	**18.0 mg/day**		
% of RDI	62.2%	73.3%	
ZINC (mg/day)	**8.5 ±0.6**	**9.5±0.5**	0.168
RDI	**11.0 mg/day**		
% for RDI	77.3%	86.4%	
CALCIUM (mg/day)	630.5 ±54.0	665.0±64.2	0.328
RDI	**1300 mg/ day**		
% of RDI	48.5%	51.2%	
PHOSPHORUS (mg/day)	765.7 ±53.2	958.7±87.9*	0.03
RDI	**1250 mg/day**		
% for RDI	61.2%	76.7%	
MAGNESIUM (mg/day)	116.9 ±11.3	123.9±14.7	0.359
RDI	**420 mg/day**		
% from RDI	27.8%	**29.5**	
POTASSIUM (mg/day)	1797.3±129.8	2038.1±140.3	0.121
RDI	**4700 mg/day**		
% of RDI	38.2%	43.4%	
SODIUM (mg/day)	2824.7±274.5	3569.8±316.3	0.05
RDI	**2300 mg/day**		
% from RDI	122.8%	155.2%	

**The mean difference is Significant at P < 0.05. RDI according to FDA 202 Intake levels of 75- 100 percent of the RDIs are regarded as adequate, whereas levels of less than 50 percent of the RDIs are deemed insufficient.*

Table 5. Distribution of kidney function tests (mean ± SD), and eGFR% in all studied groups.

Parameters	Normal range	G1 normal group	G2 patient group	P value
Urea (mg /dl)	**5 - 20**	19.5 ±1.3	86.5±6.5*	**0.000**
Creatinine (mg/dl)	**0.7 - 1.3**	0.9±0.02	3.8± 0.5*	**0.000**
Uric acid (mg/ dl)	**2.5 - 7.0**	4.1±0.2	7.9±0.4*	**0.00**
eGFR %	**> 90**	105.7±2.9	21.8±4.6*	**0.000**

eGFR= estimated Glomerular Filtration Rate. The mean difference is Significant at P < 0.05.

Table 6. Distribution of Serum levels of cardiac parameters CK-MB, troponin, LDH, and VCAM-1(mean ± SD), in all studied groups.

Parameters	Normal range	G1 normal group	G2 patient group	P value
CK MB (IU /L)	**5 -25**	**13.5±1.2**	**33±3.2***	**0.000**
Troponin (ng/mL)	**0 -0.04**	**0.03±0.01**	**0.5±0.1***	0.011
LDH (U/ L)	**140 - 280**	**98.9±4.2**	**190±4.4***	0.000
VCAM-1 (ng/mL)	**449-1103**	**216.35±4.5**	**391.6±7.6***	0.000

**The mean difference is Significant at P < 0.05*

CK-MB: Creatinine kinase-MB; LDH: Lactate dehydrogenase; VCAM-1 vascular cellular adhesion molecule-1.

Table 7. Distribution significance of serum Lipid profile Atherogenic indices (mean ± SD), in all studied groups.

Groups / Parameters	Normal range	G1 normal group	G2 patient group	P value
Triglycerides (mg/dl)	< 130	96.2±6.8	154.9±12.3*	0.000
Total Cholesterol (mg/dl)	< 200	153.1±11.2	172.3±2.9*	0.023
HDL-c (mg/dl)	> 60	44.6±6.3	35.7±10.9	0.001
LDL-c (mg/dl)	< 100	86.17±40.74	108.2±24.4*	0.005
VLDL (mg/dl)	2 - 30	21.19±5.58	31.59±12.1*	0.001
Atherogenic Index	0.11-0.21	0.07 ±0.02	0.67±0.02*	0.000
Atherogenic coefficient	2.02	0.9 ±0.2	4.4±0.4*	0.000

**The mean difference is Significant at P < 0.05 SD: Standard deviation HDL-c: High-density lipoprotein cholesterol. LDL-c: low-density lipoprotein cholesterol VLDL-c: very low-density lipoprotein cholesterol*

Table 8. Distribution of serum levels of vitamin D and vitamin D binding protein (DBP) (mean ± SD).

Parameters	Normal range	G1 normal group	G2 patient group	P value
Vitamin D (ng/ml)	**30 - 50**	**53.4±6.5**	**38.7±2.4***	**0.000**
VDBP (mg/l)	**200 - 600**	**214.0**	**216.5**	**0.334**

*DBP: Vitamin D binding protein * the results are significant when (P<0.05)*

المؤشرات الغذائية والبيوكيميائية لمرضى الكلى المزمنه والقلب والأوعية الدموية مقارنة بالمجموعة الطبيعية

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الملخص العربي

المرض الكلوي المزمن (CKD) ومرض أوعية القلب (CVD) يزيدان من تفاقم بعضهما البعض، مما يؤدي إلى زيادة معدلات الإصابة بالمرض والوفيات. يتطلب دراسة هذه الحالات المترابطة مراقبة تغذوية وبيوكيميائية صارمة. ذلك من خلال مقارنة تناول المواد الغذائية الكبيرة والدقيقة بالجرعة اليومية الموصى بها(RDI) ، توفر المتابعة المعلومات البيوكيميائية حول الأمراض الأيضية، مما يساعد على التشخيص المبكر وتنظيمات العلاج المخصصة. شملت هذه الدراسة 60 فرداً: 30 شخصاً صحيين و30 مريضًا يعانون من CKD و CVD من عيادة المرضى الخارجيين في المعهد القومي للمسالك البولية وأمراض الكلى في القاهرة، مصر. أظهرت النتائج أن تناول المواد الغذائية الكبيرة في كلا المجموعتين كان أقل من RDI باستثناء البروتين. كان لدى الجميع عجز في فيتامين د المتناول، حيث تقل نسبته عن 50٪ منRDI ، مع ملاحظة فروقات كبيرة في مستويات د بالدم، حيث كانت أقل في مجموعة .CKD + CVD باستثناء تناول الصوديوم، الذي كان أعلى من RDI في كلا المجموعتين، كان جميع تناول المعادن أقل منRDI ، مع تناول المغنيسيوم والبوتاسيوم أقل من 50٪ من .RDI . كانت العلامات البيوكيميائية للوظائف الكلوية، باستثناء معدل تصفية الغلوميرولي (eGFR) المقدر، أقل من الطبيعي في مجموعة المرضى. كانت لدى مجموعة المرضى تركيزات أعلى من العلامات القلبية مثل إنزيم الكرياتين كيناز، والتروبونين، وإنزيم لاكتات ديهيدروجيناز(LDH) ، وجزيء الالتصاق الخلوي الوعائي-1 .(VCAM-1) . أظهرت نتائج الدهون لديهم زيادات ملحوظة في الجليسريدات الثلاثة ، والدهون منخفضة الكثافة(LDL-C) ، والدهون منخفضة الكثافة جدا، ومؤشر الصورة الشحمية(AI) ، ومعامل الصورة الشحمية(AC) ، بينما انخفضت مستويات الدهون عالية الكثافة (HDL-C) بشكل ملحوظ. تؤكد هذه الدراسة أن التدخل التغذوي والعلامات البيولوجية الجديدة ضرورية لهؤلاء المرضى.

الكلمات المفتاحية : مرضى الكلى والقلب والأوعية الدموية المزمنة – التغذية – المعلمات الحيوية