

**Bioelectrical Impedance Analysis for Nutritional Assessment in Children  
with Chronic Kidney Disease**

**Abstract**

**Background:** Malnutrition and growth failure are major problems among children with chronic kidney disease (CKD). No single metric can describe their nutritional status; therefore, a series of indices and methods are required for evaluation.

**Aim of the Work:** Evaluation of the nutritional status of children with CKD using dietetic history, anthropometric measurements, biochemical parameters and BIA.

**Methods:** This case-control study was conducted on forty CKD children (stages 3-5) from those attending Pediatric Department, Tanta University Hospitals between March 2021 and February 2022 in comparison to forty healthy control children. All were subjected to: dietetic history, anthropometric measurements including (weight, height, body mass index, mid-arm circumference, skin fold thickness), BIA using TANITA Body Composition Analyzer MC-980 MA-N plus III device and laboratory investigations: (CBC, urea, creatinine, ABG, sodium, potassium, phosphorus, calcium, alkaline phosphatase, parathormone hormone, serum proteins, albumin and 24hrs urinary proteins).

**Results:** CKD children had significantly lower caloric intake and other nutrient consumption including protein, carbohydrate and fat intake ( $p < 0.05$ ), significantly lower anthropometric measurements including weight, height, body mass index, skinfold thickness and mid-arm circumference than controls ( $p < 0.05$ ) and significantly lower in BIA measurements including fat mass (FM), fat free mass (FFM), muscle mass (MM), total body water (TBW) and bone mass (BM) than controls. There was significant positive correlation between caloric intake and protein intake with FFM, MM, SMM, BM, TBW and BMR, also there was significant positive correlation between serum albumin and FFM, MM, SMM, BM and TBW in CKD patients

**Conclusions:** CKD children had low caloric and protein intake, low body composition parameters, so good nutritional assessment and improvement of their nutritional status is very important. BIA could be used with dietary assessment and anthropometric measurements to achieve more accurate nutritional evaluation in CKD children.

**Keywords:** Bioelectrical Impedance Analysis, Nutritional Assessment, Children, Chronic Kidney Disease.

UNDER PEER REVIEW

## **Introduction:**

Chronic kidney disease (CKD) is a clinical illness defined by a progressive decline in kidney function. In registries, congenital abnormalities of the kidney and urinary system account for around 50% of children CKD, followed by inherited nephropathies and glomerulonephritis (1).

Recent definitions of paediatric malnutrition describe it as an imbalance between dietary needs and consumption, resulting in accumulating deficiencies of energy, protein, and micronutrients that can severely impact growth, development, and other related outcomes (2). Patients with CKD are at a high risk for malnutrition, which is characterized by a loss of protein energy and vitamin deficit. The frequency of malnutrition is significant among both children and adults with CKD, according to studies. Malnutrition also contributes to the usual occurrence of stunted growth in children with CKD. The pathogenic mechanisms of malnutrition in chronic kidney disease (CKD) are complex and involve an interplay of multiple pathophysiologic alterations, including decreased appetite and nutrient intake, hormonal derangements, metabolic derangements, inflammation, increased catabolism, and dialysis-related abnormalities including protein loss, salt wasting, and disruption of the hydration state. Malnutrition increases these patients' risk of morbidity, death, and total disease burden (3).

Due to the insufficiency of dietary intervention, early identification and prevention of malnutrition are seen as crucial. To detect its symptoms as early as possible, it is vital to examine nutrition status on a regular basis using markers supported by scientific data. Compared to anthropometric measures, biochemical markers exhibit low diagnostic and prognostic accuracy in malnutrition evaluation. The body mass index (BMI) is acknowledged as a predictor of death in children with chronic kidney disease (CKD). However, its usefulness, as well as the validity of other anthropometric measures, is highly debatable,

primarily because CKD is linked to hydration status abnormalities. In measuring body composition among CKD patients, no traditional metric is regarded as better to another.

Using dietetic history, anthropometric measures, biochemical markers, and BIA, the purpose of the study was to assess the nutritional status of children with CKD.

## **Materials and Methods:**

This case-control study was carried out on 80 children and adolescents with CKD (stages 3 – 5) with age ranging from 5 to 18 years at Pediatric Department, Tanta University Hospitals between March 2021 and February 2022.

Exclusion criteria were chronic infection, neurological disease e.g. (cerebral palsy) and other systemic diseases e.g. (diabetes mellitus, hypothyroidism, Cushing syndrome).

Group I: forty children diagnosed as having CKD from those attending the Pediatric Nephrology Unit.

Group II: forty healthy children of matched age and sex served as controls.

All patients were subjected to.

### **1. History taking with special emphasis on:**

- Past history especially history regarding the age of onset and causes of CKD.
- Complete nutritional and feeding history.

### **2. Clinical Examination including:**

- Nutrition-focused physical examination.
- Blood pressure measurement.

### **3. Anthropometric measurements including:**

Weight, height, BMI, skin fold thickness and mid upper arm circumference. All measures were plotted on growth charts and z-scores calculation were done.

### **4. Laboratory Investigations:**

- Complete blood count (CBC).
- Blood urea and serum creatinine.
- Venous blood gas (venous blood pH and bicarbonate).

- Serum sodium, potassium, ionized calcium and phosphate.
- Serum alkaline phosphatase, Parathormone hormone and 25-OH vitamin D.
- Serum proteins and albumin.
- Serum cholesterol and triglycerides.
- Collection of 24 hours urine was done for calculation of 24hrs urinary proteins. Glomerular filtration rate (GFR) was calculated individually by Schwartz equation.

#### **5. Bioelectrical Impedance Analysis (BIA):**

BIA was used for assessing total body composition using (TANITA Body Composition Analyzer, MC-980 MA-N plus III, JAPAN). The device gave the values of fat mass (FM), fat free mass (FFM), muscle mass (MM), skeletal muscle mass (SMM), total body water (TBW), bone mass (BM) and basal metabolic rate (BMR) of every case and control <sup>(1)</sup>.

#### **Statistical analysis**

Data were entered into the computer and analysed using version 20.0 of the IBM SPSS software suite. (Armonk, New York: IBM Corporation) Quantitative and percentage descriptions were provided for qualitative data. The Kolmogorov-Smirnov test was performed to determine the distribution's normality. The range (minimum and maximum), mean, standard deviation, median, and interquartile range were used to characterise quantitative data (IQR). To compare categorical variables between groups, the Chi-square test, Fisher's exact test, or Monte Carlo correction was utilised. For quantitative variables with a normal distribution, the Student t-test was employed to compare two study groups. For quantitative variables with an anomalous distribution, the Mann Whitney test was employed to compare two groups. For quantitative data with an anomalous distribution, the Wilcoxon signed ranks test was employed to compare two periods. For quantitative variables with a normal distribution, the F for One-way ANOVA test was used to compare more than two groups, and the Post Hoc test (Tukey) was used for pairwise comparisons. For quantitative

variables with an atypical distribution, the Kruskal-Wallis test was used to compare more than two groups, and Post Hoc (Dunn's multiple comparisons test) was used for pairwise comparisons. Spearman coefficient was employed to determine the correlation between two improperly distributed quantitative variables. Pearson coefficient was used to determine the correlation between two quantitative variables with normal distributions. At the 5% significance level, the acquired findings were deemed significant.

## Results:

No significant difference between CKD patients and controls as regard age, sex and residence.

**Table 1: Demographic Data of the Studied Groups**

	<b>CKD (n = 40)</b>	<b>Control (n = 40)</b>	<b>Test of Sig.</b>	<b>P</b>
<b>Age (years)</b> Mean $\pm$ SD.	11.61 $\pm$ 3.36	10.95 $\pm$ 3.35	t=0.859	0.393
<b>Sex</b>				
Male	23 (57.5%)	22 (55%)	$\chi^2=$ 0.278	0.598
Female	17 (42.5%)	18 (45%)		
<b>Residence</b>				
Rural	30 (75%)	24 (60%)	$\chi^2=$ 1.930	0.165
Urban	10 (25%)	16 (40%)		

SD: Standard deviation t: Student t-test  $\chi^2$ : Chi square test\*: Statistically significant at ( $p \leq 0.05$ )

Duration of CKD in our children had median of 60 months with IQR of (24-84) months, as regard etiology of CKD among our CKD children, congenital causes (55%) were more common than acquired causes (45%) and glomerulonephritis was the most common single cause (35%), followed by hypoplastic and aplastic kidneys (25%), then obstructive uropathy (20%) and Thrombotic Microangiopathy (10%), but the least common causes were polycystic (7.5) and horseshoe kidney (2.5%), in this study there were 40 CKD children 42.5% of them were stage 5 (ESRD) and 7.5% of them were stage 4 and 50% of them were stage 3 and also our CKD children had elevated blood pressure with 15% of them were prehypertensive, 35% were stage 1 hypertension and 50% of them were stage 2 hypertension.

**Table 2: CKD Related Data in CKD Group**

<b>CKD (n=40)</b>	<b>No. (%)</b>
<b>Disease duration (months)</b>	
Median (IQR)	60.0(24.0 – 84.0)
<b>Etiology of CKD</b>	
• Congenital	22 (55%)

- Hypoplastic and aplastic	10 (25%)
- Obstructive uropathy	8 (20%)
- Polycystic kidney	3 (7.5%)
- Horseshoe kidney	1 (2.5%)
• <b>Acquired</b>	<b>18 (45%)</b>
- Glomerulonephritis	14 (35%)
- Thrombotic Microangiopathy	4 (10%)
<b>Stages of CKD</b>	
Stage 3 (GFR 30-59 ml/m <sup>2</sup> /1.73m <sup>2</sup> )	20 (50%)
Stage 4(GFR15-29 ml/m <sup>2</sup> /1.73m <sup>2</sup> )	3 (7.5%)
Stage 5 (GFR <15 ml/m <sup>2</sup> /1.73m <sup>2</sup> )	17 (42.5%)
<b>Blood pressure</b>	
➤ Elevated blood pressure (pre-Hypertension)	6 (15%)
➤ Stage1 HTN	14 (35%)
➤ Stage2 HTN	20 (50%)

CKD children had significantly lower caloric intake, protein, carbohydrate and fat intake than controls.

**Table 3: Three Days Diet Recall in the Studied Groups**

	Dietetic History	CKD group (n = 40)	Control (n = 40)	T	P
		Mean ± SD	Mean ± SD		
Caloric	Actual Total Calories (kcal/day)	899.55 ± 271.22	1826.09 ± 203.53	16.537*	<0.001*
	% Calories from EER	56.83 ± 12.49	100.71 ± 6.21	19.615*	<0.001*
Proteins	Amount (g/day)	33.98 ± 11.30	62.74 ± 10.39	11.419*	<0.001*
	% Of calories	15.12 ± 2.91	13.68 ± 1.35	2.797*	0.007*
	% Of actual intake from RDI	89.96 ± 24.81	168.29 ± 30.37	12.289*	<0.001*
CHO	Amount (g/day)	187.90 ± 51.12	271.03 ± 34.99	8.299*	<0.001*
	% Of calories	60.14 ± 4.79	59.45 ± 4.0	0.671	0.505
Fat	Amount (g/day)	38.69 ± 14.50	54.29 ± 9.86	5.504*	<0.001*
	% Of calories	27.40 ± 5.26	26.87 ± 4.08	0.476	0.635

SD: Standard deviation t: Student t-test CHO: carbohydrates

Weight, height, BMI, triceps skin fold thickness and mid upper arm circumference Z-scores were significantly lower in CKD children than controls.

**Table 4: Anthropometric Measurements in the Studied Groups**

Anthropometric measures	CKD (n = 40)	Control (n = 40)	Test of Sig.	P
	Median (IQR)	Median (IQR)		
Weight (Z-score)	-1.44(-2.9 – -0.94)	0.11(-0.29 – 0.61)	U= 182.00*	<0.001*
Height (Z-score)	-1.70(-2.7 – -1.10)	-0.14(-0.44 – 0.35)	U= 137.50*	<0.001*

BMI (Z-score)	-0.64(-1.32 – 0.16)	0.21(-0.24 – 0.90)	U= 389.50*	0.001*
MUAC (Z-score)	-1.65(-1.74 – -1.09)	-0.52(-0.67 – -0.03)	U= 101.50*	<0.001*
TSF (Z-score)	-1.04(-1.3 – -0.67)	-0.67(-1.0 – -0.19)	U= 488.00*	0.023*

IQR: Inter quartile range, U: Mann Whitney test, SD: Standard deviation: Student t-test BMI: Body mass index, MUAC: Mid-upper arm circumference, TSF: Triceps skin fold thickness.

CKD children had significantly lower values as regard hemoglobin concentration than controls but there was no significant difference between them as regard platelets and total leucocytic count and significantly higher values as regard serum cholesterol, triglycerides, phosphorus, alkaline phosphatase and parathormone hormone, CKD children had significantly lower values as regard serum proteins, albumin, ionized calcium and 25-OH Vitamin D.

**Table 5: CBC and Biochemical Nutrition Assessment in the Studied Groups**

CBC		CKD (n = 40)	Control (n =40)	Test of sig.	P
		Mean ± SD/ Median (IQR)	Mean ± SD / Median(IQR)		
Hemoglobin (gm/dl)		9.68 ± 1.17	11.54 ± 0.49	t= 9.175*	<0.001*
Platelets (×10 <sup>3</sup> /mm <sup>3</sup> )		242.8 ± 78.99	248.7 ± 60.36	t= 0.364	0.717
Total leucocytic count (×10 <sup>3</sup> /mm <sup>3</sup> )		7.10(5.2 – 8.1)	6.60(5.4 – 8.4)	U= 679.50	0.828
Differential leucocytic count	Neutrophils (%)	57.06 ± 11.10	56.63 ± 6.0	t= 0.210	0.834
	Lymphocytes (%)	29.5 (23 – 37.5)	28.0(24.0 – 30.0)	U= 592.50	0.253
Total proteins (g/dl)		6.29 ± 0.88	7.41 ± 0.41	t= 7.245*	<0.001*
Serum albumin (g/dl)		3.52 ± 0.51	4.23 ± 0.29	t= 7.516*	<0.001*
Serum cholesterol (mg/dl)		178.97 ± 47.29	148.73 ± 17.83	t= 3.752*	<0.001*
Serum triglycerides (mg/dl)		134.7 ± 39.64	98.27 ± 14.18	t= 5.421*	<0.001*
Serum ionized calcium (mg/dl)		1.03 ± 0.12	1.16 ± 0.07	t= 5.757*	<0.001*
Serum phosphorus (mg/dl)		4.79 ± 0.87	4.37 ± 0.41	t= 2.735*	0.008*
Serum alkaline phosphatase (IU/L)		207.0 ± 82.40	123.1 ± 22.76	t= 6.174*	<0.001*
Serum parathormone hormone (Pg/ml)		166(97.5 -224.0)	24(20.9 – 26.5)	U= 40.0*	<0.001*
25-OH Vitamin D (ng/ml)		18.95(15.8 – 24.3)	28.60(26.0 – 31.4)	U= 225.0*	<0.001*

IQR: Inter quartile range U: Mann Whitney test SD: Standard deviation t: Student t-test.

CKD children had significantly higher values as regard blood urea, serum creatinine, potassium, and 24 hrs urinary proteins than controls, CKD children had significantly lower values as regard serum sodium, PH, HCO<sub>3</sub> and GFR than controls.

**Table 6: Renal and Metabolic Investigations in the Studied Groups**

	CKD (n = 40)	Control (n = 40)	Test of sig.	P
	Mean ± SD/ Median (IQR)	Mean ± SD / Median (IQR)		
Blood urea (mg/dl)	68 (56.5 -106.0)	27 (22 – 32.5)	U=0.500*	<0.001*
Serum creatinine (mg/dl)	1.75(1.2 – 6.0)	0.60(0.50 – 0.75)	U=4.0*	<0.001*
Serum sodium (mEq/L)	137.3 ± 4.16	140.5 ± 2.49	t=4.129*	<0.001*
Serum potassium(mEq/L)	4.85 ± 0.60	4.24 ± 0.50	t=4.678*	<0.001*
PH (mmol/L)	7.35 ± 0.04	7.40 ± 0.03	t=6.626*	<0.001*
HCO <sub>3</sub> (mmol/L)	17.67 ± 2.58	24.35 ± 1.22	t=14.575*	<0.001*
24 hrs urinary proteins (mg/m <sup>2</sup> /hr)	8 (5.5 – 16.0)	0.70(0.50 – 1.1)	U=0.00*	<0.001*
GFR (ml/m <sup>2</sup> /1.73m <sup>2</sup> )	46(11.8 –52.7)	122(114.5 – 137.3)	U=0.00*	<0.001*

IQR: Inter quartile range, U: Mann Whitney test, SD: Standard deviation, t: Student t-test.

CKD children were significantly lower in body composition parameters (FM, FFM, MM, TBW, BM and BMR) than controls but no significant difference between them as regard SMM.

**Table 7: BIA Parameters in the Studied Groups**

BIA	CKD (n = 40)	Control (n = 40)	Test of sig.	P
FM (Kg)	6.42 ± 2.19	8.97 ± 3.99	t=3.358*	0.001*
FFM (Kg)	23.10(18.75 – 30.05)	26.40(21.85 – 36.10)	U=495.50*	0.030*
MM (Kg)	21.80(17.65 – 28.65)	24.90(20.70 – 34.25)	U=494.0*	0.029*
SMM (Kg)	12.90(10.50 – 17.0)	13.20(11.35 – 17.0)	U=608.0	0.328
BM	1.20(1.0 – 1.60)	1.40(1.20 – 1.85)	U=491.0*	0.026*
TBW (Kg)	16.80(13.65 – 23.35)	19.30(16.15 – 26.05)	U=509.50*	0.043*
BMR	1142.93 ± 199.76	1231.43 ± 166.53	t=2.078*	0.041*

IQR: Inter quartile range, U: Mann Whitney test, SD: Standard deviation t: Student t-test FM: Fat mass FFM: Fat free mass, MM: Muscle mass SMM: skeletal muscle mass TBW: total body water BM: bone mass.

BMR: Basal metabolic rate

There was no statistically significant correlation between body composition and 24hrs urinary proteins, GFR and duration of CKD but there was significant positive correlation between FFM, MM, SMM, BM and TBW and caloric intake and also protein intake.

**Table 8: Correlation between BIA Parameters and Renal Data and Dietary Intake in CKD group**

BIA	24 hours urinary proteins		GFR		Duration of CKD		Caloric Intake(kcal/day)		Protein Intake(g/day)	
	r <sub>s</sub>	P	r <sub>s</sub>	P	r <sub>s</sub>	P	R	P	r <sub>s</sub>	P
<b>FM (Kg)</b>	-0.056	0.782	-0.192	0.235	0.203	0.208	0.183	0.258	0.261	0.104
<b>FFM (Kg)</b>	0.145	0.472	0.238	0.139	-0.310	0.052	0.388	0.013*	0.457	0.003*
<b>MM (Kg)</b>	0.220	0.270	0.027	0.867	-0.025	0.879	0.783	<0.001*	0.879	<0.001*
<b>SMM (Kg)</b>	0.233	0.242	0.019	0.907	-0.016	0.920	0.780	<0.001*	0.876	<0.001*
<b>BM</b>	0.220	0.271	-0.045	0.785	-0.092	0.572	0.796	<0.001*	0.874	<0.001*
<b>TBW (Kg)</b>	0.218	0.274	0.028	0.866	-0.022	0.895	0.726	<0.001*	0.833	<0.001*

rs: Spearman coefficient r: Pearson coefficient

There was significant positive correlation between FFM, MM, SMM, BM and TBW and serum albumin but there was no statistically significant correlation between body composition and levels of 25-OH vitamin D, ionized calcium, phosphorus, parathormone hormone, cholesterol, triglycerides and alkaline phosphatase in CKD patients.

**Table 9: Correlation between BIA Parameters and Biochemical Nutrition Assessment Parameters in CKD group**

BIA	25-OH Vitamin D		Serum albumin		Ionized calcium		Phosphorus		Parathormone hormone		Cholesterol		Triglycerides		Alkaline phosphatase		
	r <sub>s</sub>	P	r <sub>s</sub>	P	r <sub>s</sub>	P	r <sub>s</sub>	P	r <sub>s</sub>	P	r	P	R	P	r	P	
<b>FM (Kg)</b>	0.030	0.853	0.032	0.844	-0.026	0.872	0.184	0.256	0.244	0.129	0.223	0.167	0.171	0.291	-0.165	0.309	<b>0.309</b>
<b>FFM (Kg)</b>	-0.013	0.936	0.368*	0.020*	-0.193	0.232	0.047	0.771	0.205	0.204	-0.130	0.425	-0.018	0.913	-0.063	0.700	<b>0.700</b>
<b>MM (Kg)</b>	-0.035	0.829	0.367*	0.020*	-0.192	0.236	0.049	0.764	0.209	0.195	0.089	0.585	0.129	0.428	-0.226	0.161	<b>0.161</b>
<b>SMM (Kg)</b>	-0.054	0.742	0.376*	0.017*	-0.187	0.248	0.047	0.774	0.189	0.243	0.093	0.568	0.139	0.393	-0.243	0.131	<b>0.131</b>
<b>BM</b>	-0.080	0.625	0.332*	0.036*	-0.256	0.111	0.053	0.745	0.175	0.280	0.044	0.788	0.107	0.512	-0.229	0.155	<b>0.155</b>
<b>TBW (Kg)</b>	-0.051	0.756	0.406*	0.009*	-0.165	0.309	0.018	0.914	0.211	0.190	0.109	0.504	0.137	0.399	-0.219	0.175	<b>0.175</b>

rs: Spearman coefficient r: Pearson coefficient

## Discussion

The nutritional condition of children with chronic kidney disease (CKD) is complicated due to the fluctuating needs for maximising both physical and cognitive growth during childhood. Therefore, it is essential for the physician to have appropriate knowledge of nutritional status evaluation techniques (2). The optimal approach for evaluating the nutritional status of children with CKD has not been determined, as nutritional status is a challenging concept to define, and no one assessment can accurately reflect nutritional status (3).

The intake of energy, protein, fat, and carbohydrate was determined by analysing the 3-day dietary recall records, which revealed a general decrease in intake of all constituents in patients compared to controls. This is consistent with the findings of Gupta et al (4), who found that the average dietary intake of calories was lower in all stages of CKD.

The Kidney Disease Outcomes Quality Initiative (KDOQI) recommends providing 100–140% of the DRI of protein for optimal bodyweight to children with stages 2-3 CKD, whereas children with more advanced CKD should get 100–120% of the DRI (2).

In the present study, protein intake showed significant reduction in CKD patients than controls as regard amount of protein intake but as regard percent of proteins in diet we found that control group was lower than CKD group as healthy children had good quality protein but CKD children had poorer quality plant sources, also we found that healthy controls had higher protein intake from RDI as recent advances suggests protein requirement as much as 60% higher than current recommendations (3). Our research showed that the average protein and calorie intake of the sick group, as determined by questionnaire, did not vary from that of the control group.

In fact, the children with CKD had a diminished appetite and a restricted diet. In addition, it is possible that after diagnosis, some patients are unable to limit the quality and quantity of nutrients, such as protein, fat, and vitamins, even without nutritional training and dietary intervention associated with CKD, so that not all of them are able to reach the healthy dietary

goal and the earliest symptom of malnutrition in CKD children is a decreased diet. In CKD, many factors associated with inadequate nutrient intake contributed to malnutrition such as acidosis, anaemia and hormonal imbalance <sup>(4)</sup>.

The present study revealed that weight, height, triceps skinfold thickness and mid upper arm circumference measurements and their corresponding z-scores of all children with CKD were significantly lower than those of healthy children and this is in agreement with **Yilmaz et al** <sup>(10)</sup> study.

The height was more affected than weight in CKD children in the present study with the median value of height was -1.7 and weight was -1.44 and this is in agreement with **Zhang et al** <sup>(5)</sup> study who found that height mean was  $-4.2 \pm 2.3$  SDS and the body weight mean was  $-2.84 \pm 2.38$  SDS, so that the height was the most affected anthropometric parameter in our study as 42.5% of our CKD children had ESRD, and our CKD children had acidosis, decreased ionized calcium and vitamin D levels that lead to MBD.

In the current study, the BMI mean value in CKD children and its z-score were significantly lower than controls ( $P < 0.05$ ) and this is not in agreement with **Zhang et al** <sup>(6)</sup> study which found that BMI did not differ between the patient group and the control group and he considered that BMI affected by hypervolemia is not a very reliable method to assess malnutrition in children with CKD.

As stated in the KDOQI recommendations, serum albumin may be insensitive to acute changes in nutritional status because to its lengthy half-life. Consequently, serum albumin remains a useful marker for the overall evaluation of children with CKD. In addition, serum albumin is diminished in the presence of volume excess and systemic inflammation <sup>(5)</sup>.

In our study, the mean range of level of serum albumin in CKD children was significantly lower than controls ( $P < 0.001$ ). This agrees with **Gupta et al** <sup>(6)</sup> study which found that the

mean serum albumin of the study population was  $3.60 \pm 0.38$  g/dl due to higher degree of malnutrition, lower protein intake and proteinuria.

In the present study, we found that GFR was significantly lower and 24-hours urinary proteins were significantly higher in CKD children than controls this is due to kidney injury and damage of the kidney filters that leads to proteinuria.

In the current study, we found that CKD children had increased levels of cholesterol and triglycerides as compared with controls and this agrees with **Zhang et al**<sup>(5)</sup> study which found that CKD children had increased cholesterol and triglycerides level.

Metabolic acidosis which could be an important cause for growth failure was present in our CKD children with the mean range of  $\text{HCO}_3$   $17.67 \pm 2.58$  mEq/L, while <sup>(7)</sup> found that 74% of patients had bicarbonate values above 22 mEq/L and this study had also concluded that bicarbonate values below 18 mEq/L were associated with poor height SDS scores.

We found that CKD children had decreased serum level of sodium and increased serum level of potassium with significant difference between them and controls, while **Gupta et al** study <sup>(6)</sup> found that serum sodium values were not significantly different but there was a significant increase in serum potassium in higher stages of CKD children than controls this is due to decreased ability of kidneys of CKD patients to maintain balance of water and electrolytes and as the kidney function gets worse, they may not be able to remove excess potassium from body.<sup>(8)</sup>

As regard calcium panel, we found that serum ionized calcium and serum vitamin D were significantly lower while serum phosphorus, alkaline phosphatase and parathormone hormone were significantly higher in CKD children than controls. This is in agreement with **Gupta et al study**<sup>(6)</sup> who found that the mean serum phosphates were higher and there was a significant decline in serum calcium levels in CKD children and also **Solarin et al study**<sup>(9)</sup> found that suboptimal Vitamin D levels are prevalent in children with CKD, especially those

on chronic dialysis and the mean PTH values increased with increasing CKD stage, this is explained by reduced renal function on bone and mineral (calcium and phosphate) metabolism as there is reduced renal excretion of phosphate and impaired gastrointestinal and renal reabsorption of calcium, resulting in hyperphosphatemia and hypocalcaemia leading to secondary hyperparathyroidism,<sup>(10)</sup> also vitamin D deficiency is due to reduced capacity of kidney to activate vitamin D.<sup>(11)</sup>

Regarding the use of BIA to assess malnutrition in CKD patients, there were differing opinions. Edefonti et al. (13) revealed that BIA was more sensitive than anthropometric measures for detecting and monitoring malnutrition in children undergoing automated peritoneal dialysis (APD). Reduced BIA FM was related with higher mortality risk in HD patients, as demonstrated by Segall et al. Hou et al (14) have demonstrated that BIA for assessing body compartments in HD patients contains some mistakes. KDOQI recommends BIA for assessing haemodialysis patients' body composition<sup>(2)</sup>.

In the present study, we found that there was significant difference between CKD children and controls in BIA, CKD children were significantly lower in FM, FFM, MM, TBW, BM and BMR than controls, but there was no significant difference between them as regard SMM. While Švigelj et al<sup>(12)</sup> study found that TBW was significantly higher in children with CKD than the control group, other parameters were in lower ranges compared to the control group.

Body composition parameters in CKD children was lower than healthy individuals, this was mainly due to decreased appetite, decreased caloric and protein intake and muscle wasting, also decreased bone mass is mainly due to disturbance in calcium and phosphorus metabolism and decrease vitamin D level that lead to MBD.<sup>(13)</sup>

As regard dietetic history, in our study we found that there was significant positive correlation between total caloric intake and FFM, MM, SMM, BM and TBW and also, they

had significant positive correlation with protein intake. This is in agreement with **Apostolou et al** <sup>(13)</sup> study which found significant positive correlation between body composition parameters and caloric intake and protein intake.

Limitations: The relatively small number of patients enrolled in the study and children below 5 years could not be included in our study as our BIA device could not measure their body composition.

### **Conclusions:**

CKD had negative impact on growth of CKD children, Children with CKD had lower caloric and protein intake, lower body composition parameters than healthy controls, Body composition monitoring through BIA considered as a noninvasive, easy and bedside method for assessment of the body composition that gives a better idea regarding the nutritional status of the children with CKD.

### **Ethical Approval and Consent:**

The study was approved by “ethical committee of Faculty of Medicine, Tanta University” (registration No. 34412/1/21). Participation in the study was voluntary after informed consent or assent form was obtained from each parents of the patient before enrolment in the study.

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