

Original Article

Dietary management of haemodialysis patients with chronic kidney disease and malnourishment

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Background and Objectives: Dietary supplementation for haemodialyzed (HD) patients with chronic kidney disease (CKD) and its benefits for the anthropometric profiles remain contentious. This study analysed changes in the albumin levels and anthropometric profiles of HD patients within 3 months of nutritional therapy. **Methods and Study Design:** Sixty-three malnourished HD patients (Subjective Global Assessment nutrition status B or C) were enrolled. Twenty patients received counselling, 17 patients received oral therapy, 26 patients received intradialytic parenteral nutrition (IDPN), and were evaluated at month 0, month 1, and month 3. Five patients withdrew before completing the trial. The patients' albumin levels and anthropometric profiles (biceps and triceps skinfold thickness, upper arm circumference, body weight, and body mass index) were analysed before and after treatment. We performed multivariate analysis to determine the effect of each treatment on serum albumin and anthropometric profiles. **Results:** At months 1 and 3, nutritional therapy was associated with different mean serum albumin level among three nutritional intervention groups ($p < 0.05$). Significant increases in serum albumin, upper arm circumference, and triceps and biceps skinfold thickness were identified in the counselling and IDPN groups. Multivariate linear regression revealed significant differences between oral and nonoral groups in albumin and biceps and triceps skinfold thickness at months 1 and 3. These variables were affected by age and duration of haemodialysis ($p < 0.05$). **Conclusions:** Nutritional therapy for malnourished CKD patients receiving HD ameliorated serum albumin and their anthropometric profiles within 3 months.

Key Words: albumin, nutrition, health, chronic kidney disease, haemodialysis

INTRODUCTION

Protein–energy malnutrition often occurs in patients who undergo haemodialysis (HD). Protein–energy malnutrition in chronic hemodialysis patients can be identified using several biomarkers, one of them is albumin. Albumin is often used to evaluate HD patients because it reflects decreases in visceral protein stores and can be used to predict disease progression in patients with chronic kidney disease (CKD).^{1,2} Several studies have reported a strong relationship among low serum albumin, morbidity, and mortality in HD patients.^{1,3-5}

Persistent inflammation in patients with CKD reduces the synthesis and half-life of serum albumin, thereby reducing patients' plasma protein levels. HD can lead to inadequate nutritional intake due to decreased appetite and increased dietary restrictions. Hypoalbuminaemia in an HD patient with CKD can induce hemodilution.^{2,6} Therefore, dietary adjustments are necessary to maintain serum albumin levels in the pre- and post-dialysis periods. When a patient's albumin is increased through diet, the patient's risk of mortality or morbidity is expected to decrease.^{4,7-10} However, few specific strategies for im-

proving serum albumin in HD patients have been developed.

Intradialytic parenteral and oral nutrition are commonly used for the dietary management of HD patients. Nevertheless, the clinical, anthropometric, and serum albumin-related benefits of these dietary management strategies remain contentious. This study evaluated changes in serum albumin and anthropometric profiles in HD patients after dietary adjustment.

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METHODS

Data collection

We calculated the sample size by comparative study and obtained an estimated sample size of 60. We enrolled 63 HD patients with CKD and various comorbidities, such as diabetes mellitus, hypertension, lupus, and urinary stones. Prior to study participation, patients provided informed consent. Each patient was assigned to and administered one of three types of nutritional therapy—counselling (20 patients), oral nutrition (17 patients), or intradialytic parenteral nutrition (26 patients) for 3 months. The patients' albumin, anthropometric profiles, and nutritional status were examined at baseline and at months 1 and 3.

The diets prescribed to the patients consisted of specified solid foods, meat side dishes, vegetable side dishes, vegetables, fruit, and milk. Subjective Global Assessment (SGA) was used to evaluate the patients' weight changes, dietary intake, gastrointestinal symptoms, and functional capacities as well as to ensure that each patient's SGA nutrition status was B or C.

Of the 63 patients, 5 (2 who received intradialytic parenteral nutrition therapy and 3 who received counselling therapy) withdrew from the study. The remaining 58 pa-

tients completed all 3 months of nutritional therapy (Figure 1).

Ethical declaration

Ethical clearance was provided by the Research Ethics Committee of Dr. Soetomo General Teaching Hospital (No: 0090/KEPK/XI/2020). All patients signed an informed consent agreement prior to the study.

Statistical analysis

We used SPSS version 24.0 software (Chicago, IL, USA) to conduct statistical analyses. The descriptive statistics presented herein comprise categorical variables presented as numbers (percentages) and continuous variables presented as means (\pm standard deviations) or medians (with ranges) depending on whether the data was normally distributed. We used listwise deletion or univariable and multivariable analysis to account for missing data. A Mann–Whitney U test, t test, chi-square test, or Fischer's exact test was conducted according to the type of variable analysed. Statistical significance was assessed using a chi-square for dichotomous variables and a paired-sample t test or Wilcoxon test for continuous variables depending

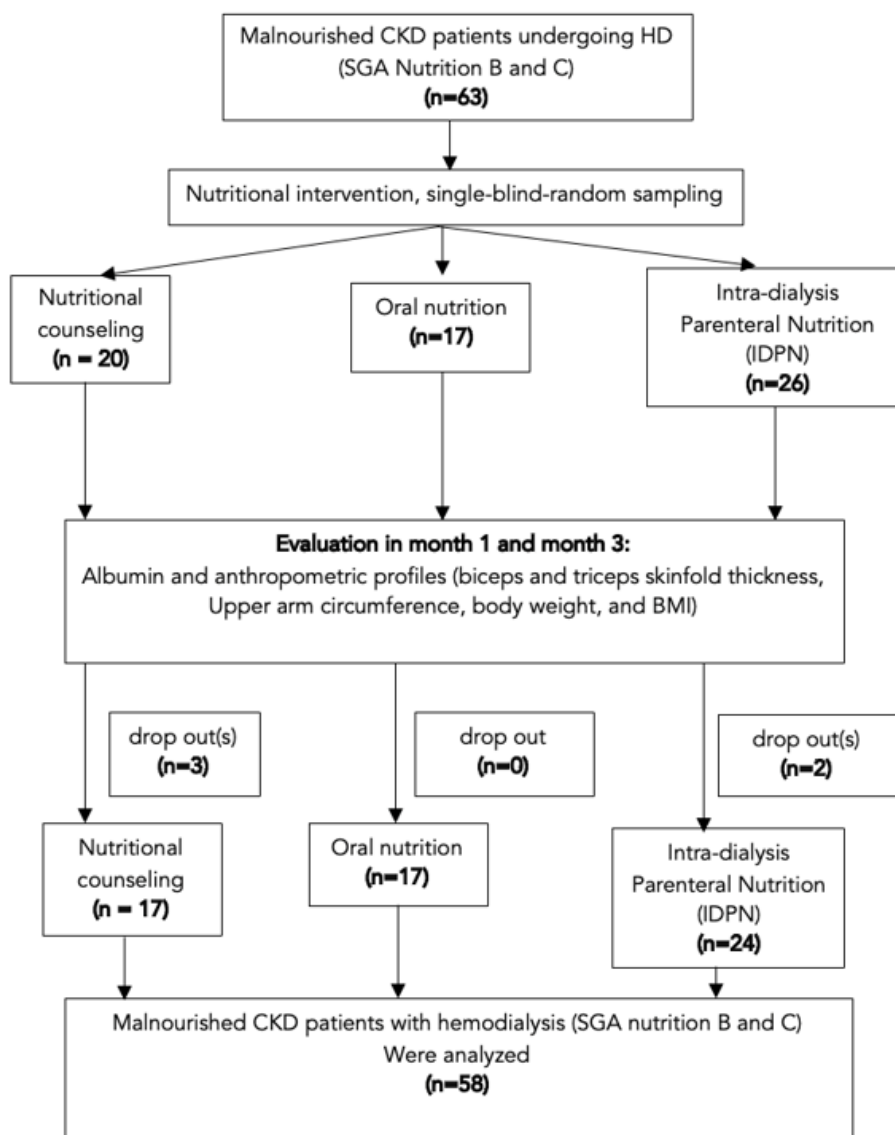


Figure 1. Flowchart of selection of haemodialysis patients with chronic kidney disease and malnourishment. CKD: chronic kidney disease; SGA: subjective global assessment; BMI: body mass index.

on whether the data was normally distributed. Analysis of variance was conducted to compare the average serum albumin and anthropometric values among the nutritional intervention groups.

Nutritional interventions were hypothesised to affect the patients' serum albumin and anthropometric parameters, and this hypothesis was assessed using a linear regression model. The nutritional interventions were categorised as oral/nonoral and IDPN/non-IDPN. This linear regression model was used for the analysis of age and duration of HD as confounding factors.

RESULTS

The 58 eligible patients were divided into three nutritional intervention groups; 17 received counselling, 17 received oral intervention, and 24 received IDPN. The study sample mostly comprised men (51.72%), and the average age was 47.4±9.63 years. The study participants were patients who had been undergoing chronic haemodialysis for an average duration of approximately 47.5±30.91 weeks.

Most of the patients had hypertension (67.24%), and many had diabetes mellitus (32.76%). The frequency of meals was a factor affecting malnutrition in the patients with end-stage renal disease. On average, the patients ate 2–3 times per day. According to the anthropometric measurements taken at baseline, significant differences in triceps fold thickness ($p<0.05$) and biceps fold thickness

($p=0.002$) between the post- and pre-intervention stages were identified in all groups. According to the patients' laboratory results, differences in serum creatinine ($p=0.01$) were identified in all groups (see Table 1).

Between months 1 and 3, the counselling group exhibited significant differences in upper arm circumference (from $p=0.009$ to $p=0.015$), triceps fold thickness (from $p=0.007$ to $p=0.01$), biceps fold thickness (from $p=0.015$ to $p=0.017$), and albumin (from $p<0.05$ to $p=0.001$). The oral intervention group exhibited a significant difference in albumin ($p=0.026$) between months 1 and 3. Comparative analysis of the patients' albumin levels and anthropometric profiles pre- and post-IDPN (at months 1 and 3) revealed a satisfactory increase in average serum albumin ($p=0.002$). Furthermore, the patients also exhibited satisfactory increases in upper arm circumference ($p=0.037$), triceps fold thickness ($p=0.011$) after IDPN intervention (Table 2).

We conducted a multivariate analysis of the variables that exhibited significant differences. The results revealed that age and duration of HD were significantly associated with serum albumin and biceps and triceps skinfold thickness in the oral nutrition group. Body mass index (BMI), age, and duration of HD were identified as confounding variables that must be controlled for. In the comparison between the IDPN and non-IDPN groups, albumin was significantly affected by age and duration of HD only in month 1. The other anthropometric param-

Table 1. Baseline characteristics of malnourished haemodialyzed patients receiving nutritional therapy

	Nutritional Intervention						<i>p</i> value	All	
	Counselling n=17		Oral n=17		IDPN n=24			n=58	
Sex							0.55		
Male (n, %)	7.00	41.2	9.00	52.9	14.0	58.3		30.0	51.7
Female (n, %)	10.0	58.8	8.00	47.1	10.0	41.7		28.0	48.3
Age (mean/SD)	46.4	12.4	51.4	6.10	45.3	8.93	0.12	47.4	9.63
Duration Haemodialysis	54.7	32.8	36.8	28.1	49.8	30.7	0.22	47.5	30.9
Comorbid									
DM (n, %)	4.00	23.5	15.0	88.2	0.00	0.00	<0.05	19.0	32.8
HT (n, %)	11.0	64.7	8.00	47.0	20.0	83.3	0.07	39.0	67.2
SLE (n, %)	1.00	5.88	0.00	0.00	0.00	0.00	0.29	1.00	1.72
Kidney Stone (n, %)	3.00	17.7	1.00	5.88	5.00	20.8	0.41	9.00	15.5
Tuberculosis (n, %)	2.00	11.8	2.00	11.8	1.00	4.17	0.60	5.00	8.62
Nephrotic syndrome (n, %)	1.00	5.88	1.00	5.88	0.00	0.00	0.48	2.00	3.45
Meal frequency							0.40		
1x (n, %)	0.00	0.00	2.00	11.8	2.00	8.33		4.00	6.90
2x (n, %)	7.00	41.2	11.0	64.7	12.0	50.0		30.0	51.7
3x (n, %)	10.0	58.8	5.00	29.4	10.0	41.7		25.0	43.1
Anthropometric profile (mean/SD)									
Weight (kg)	59.4	13.3	54.9	8.80	59.7	10.5	0.35	58.2	11.0
Height (cm)	157	7.99	162	5.89	161	7.39	0.08	160	7.38
Body mass index	24.1	5.31	20.9	3.62	23.2	4.39	0.10	22.8	4.59
Upper arm circumference (cm)	23.9	3.67	25.9	3.80	25.3	3.96	0.33	25.1	3.84
Triceps skinfold thickness (cm)	3.28	1.10	10.5	5.50	8.01	4.23	<0.05	7.39	4.92
Bicep skinfold thickness (cm)	2.57	0.68	6.55	4.46	4.52	3.04	0.00	4.54	3.44
Laboratory (mean/SD)									
Hb (g/dL)	10.1	2.06	9.43	1.57	9.33	1.66	0.38	9.58	1.76
Serum iron (mcg/dL)	65.4	22.5	60.5	34.4	61.3	25.9	0.86	62.2	27.4
TIBC (mcg/dL)	233	44.7	240	88.5	209	101	0.46	225	84.1
Albumin (g/dL)	3.40	0.17	3.37	0.22	3.46	0.20	0.37	3.41	0.19
BUN (mg/dL)	67.7	12.5	59.6	14.1	71.1	27.7	0.21	66.7	20.8
Creatinine serum (mg/dL)	11.2	2.62	9.65	2.88	12.3	2.57	0.01	11.2	2.86

DM: diabetes mellitus; HT: hypertension; SLE: systemic lupus erythematosus; Hb: hemoglobin; TIBC: total iron-binding capacity; BUN: blood urea nitrogen.

Table 2. Outcomes evaluated at months 1 and 3 after nutritional interventions

	Counselling (n=17)					Oral (n=17)					IDPN (n=24)					
	Month 1		Month 3		<i>p</i> value	Month 1		Month 3		<i>p</i> value	Month 1		Month 3		<i>p</i> value	
Anthropometric profile [†]																
Weight (kg) (mean; SD)	58.4	10.8	58.4	10.8	0.86	55.1	8.64	54.9	8.22	0.57	59.8	10.5	60.0	10.6	0.65	
Body Mass Index (mean; SD)	24.2	5.23	24.3	5.25	0.81	21.0	3.58	20.9	3.49	0.59	23.3	4.41	23.4	4.43	0.65	
Upper arm circumference (cm; mean; SD)	23.9	3.67	25.0	4.14	0.01*	25.9	3.79	26.0	3.65	0.39	25.3	3.96	25.4	3.54	0.69	
Triceps skinfold thickness (cm; mean; SD)	3.28	1.10	3.86	0.95	0.01*	10.5	5.52	11.1	5.92	0.34	8.10	4.21	8.69	4.92	0.13	
Biceps skinfold thickness (cm; mean; SD)	2.58	0.68	2.97	0.76	0.02*	6.55	4.46	7.21	4.38	0.24	4.51	3.04	4.64	2.78	0.69	
Laboratory [†]																
Albumin (g/dL; mean; SD)	3.43	0.19	3.52	0.21	<0.05	3.31	0.26	3.37	0.26	0.03	3.50	0.22	3.56	0.29	0.02	
Improvement Anthropometric profile [†]																
Weight increase (kg; n; %)	9.00	52.9	12.0	70.6	0.52	8.00	47.1	10.0	58.8	0.80	14.0	58.3	15.0	62.5	0.22	
Body mass index increase (n; %)	9.00	52.9	12.0	70.6	0.49	8.00	47.1	10.0	58.8	0.80	14.0	58.3	15.0	62.5	0.18	
Upper arm circumference increase (cm; n; %)	8.00	47.1	12.0	70.6	0.02*	6.00	35.3	7.00	41.2	0.39	7.00	29.2	11.0	45.8	0.04*	
Triceps skinfold thickness increase (cm; n; %)	8.00	47.1	12.0	70.6	0.01*	8.00	47.1	10.0	58.8	0.26	14.0	58.3	18.0	75.0	0.01*	
Biceps skinfold thickness increase (cm; n; %)	8.00	47.1	12.0	70.6	0.02*	8.00	47.1	10.0	58.8	0.30	14.0	58.3	16.0	66.7	0.11	
Laboratory [‡]																
Albumin increase (g/dL; n; %)	7.00	41.1	14.0	82.4	0.00*	4.00	23.5	6.00	35.3	0.22	12.0	50.0	14.0	58.3	0.002	

IDPN: Intradialytic Parenteral Nutrition.

[†]paired-sample *t* test.[‡]Wilcoxon test, and ⁺*p* value <0.05.**p*<0.05 (Independent sample *t* test and multiple linear regression conducted using age and duration of haemodialysis as independent variables).

ters, as well as albumin, were not significantly different among the groups after 3 months of nutritional intervention (Table 3 and Figure 2).

DISCUSSION

In the United States, approximately 400,000 individuals with stage 5 CKD undergo chronic HD (CHD) per year, with an average mortality risk of 20%–25%. Despite cardiovascular disease being a major cause of death, conventional cardiovascular risk factors such as hypercholesterolaemia or hypertension do not strongly affect mortality in patients with CKD. However, low serum albumin, poor protein intake, and low BMI or weight loss are strong predictors of mortality in CHD patients.¹²

Patients with CKD are prone to experiencing malnutrition or protein energy wasting (PEW) due to several factors. CKD requires patients to follow a low-protein diet (0.8 g/kg body weight) to prevent uraemia, and chronic inflammation causes PEW. Furthermore, CHD leads to intradialytic loss of amino acids and albumin.^{13,14}

Because PEW is related to mortality risk in patients with CKD, interventions that alter a patient’s nutritional status can improve their chance of survival. Hypoalbuminaemia is the most common marker of PEW in dialysis patients and is strongly related to mortality. The use of IDPN is a prospective strategy to correct PEW-related conditions, especially intradialytic hypoalbuminaemia.¹²

Goldstein et al. demonstrated that IDPN is an effective treatment for organic causes of PEW in adolescent and young adult patients undergoing CHD. The main advantage of IDPN is that it provides 37%–42% of protein intake, whereas only 10% of a patient’s protein intake may originate from their total recommended weekly ca-

loric intake.¹³

Capelli et al. revealed that a treatment group exhibited an increase in body weight after 8 months (157±40 lbs) of therapy; body weight continued increasing through the 12th month (169±44 lbs).¹⁵

Another study conducted in Los Angeles reported an increase in serum albumin in IDPN recipients, who comprised 72% of the patients in the study. Among these patients, 59% exhibited an increase in serum albumin of 0.5 g/dL or more. Demographic analysis revealed a significant difference in serum albumin levels from the beginning of the study, indicating that 37% of the respondent patients were in a state of severe hypoalbuminaemia (<3.0 g/dL) before receiving IDPN. Our study revealed that among the three nutritional intervention groups in the first month, the IDPN group exhibited the highest increase in albumin; however, after the third month, the counselling group exhibited the highest increase in serum albumin (3.4 to 3.52), whereas the average albumin of the IDPN group remained comparatively stable (3.5 to 3.56).¹²

The logistic regression analysis (controlled for age, sex, diabetes mellitus, and IDPN time) indicated that the odds ratio (OR) of IDPN was 85% higher for each 0.5 g/dL increase in albumin. After dichotomising the serum albumin at 3.0 g/dL, the possibility of a response to IDPN was 2.5 times higher in the patients with severe hypoalbuminaemia than in those without it (95% confidence interval [CI]: 1.3–4.9, *p*=0.006). Additional multivariate logistic regression analysis adjusted for the same covariates revealed that the likelihood of an increase in serum albumin of at least 0.5 g/dL during IDPN therapy was 3.5 times higher in the patients with severe hypoalbuminaemia than in those without it (95% CI: 1.8–6.8, *p*<0.001).¹²

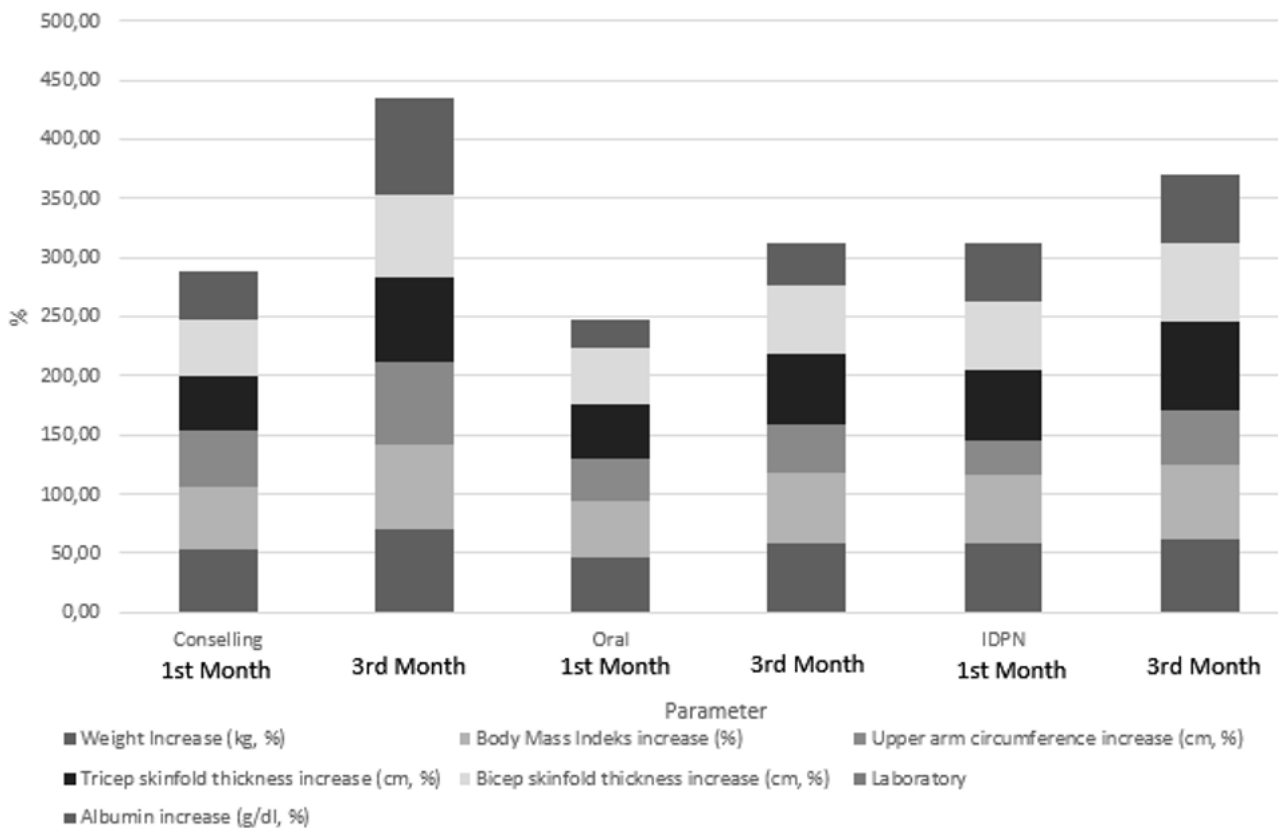


Figure 2. Improvements in serum albumin and anthropometric profiles at 1 and 3 of nutritional therapy.

Table 3. Outcomes evaluated at months 1 and 3 after nutritional interventions[†]

Month 1	Anthropometric profile														
	Weight (kg)			Body mass index			Upper arm circumference			Triceps skinfold thickness			Biceps skinfold thickness		
	Mean	MD (95%CI)	<i>p</i> value	Mean	MD (95%CI)	<i>p</i> value	Mean	MD (95%CI)	<i>p</i> value	Mean	MD (95%CI)	<i>p</i> value	Mean	MD (95%CI)	<i>p</i> value
Unadjusted [‡]															
Oral	55.1	4.60 (-10.2-0.92)	0.10	21.0	2.71 (0.40-5.01)	0.02*	25.9	1.16 (-1.08-3.4)	0.30	10.5	4.38 (1.32-7.45)	0.01*	6.55	2.84 (0.44-5.24)	0.02*
Non Oral	59.7			23.7			24.7			6.10			3.71		
IDPN	59.8	2.47 (-3.2-8.28)	0.39	23.3	0.69 (-1.74-3.11)	0.57	25.3	0.35 (-1.70-2.43)	0.74	8.10	1.22 (-1.30-3.75)	0.34	4.51	0.05 (-1.83-1.74)	0.96
Non IDPN	57.4			22.6			24.9			6.88			4.56		
Adjusted [§]															
Oral	55.10	3.08 (-10.8-1.54)	0.14	21.0	2.71 (0.50-5.27)	0.04**	25.5	0.62 (-1.79-3.04)	0.61	10.2	3.99 (1.16-6.81)	0.01*	6.37	2.58 (0.55-4.62)	0.01*
Non Oral	59.7			23.7			24.8			6.22			3.79		
IDPN	59.5	2.95 (-3.9-7.84)	0.51	23.2	0.49 (-2.03-3.00)	0.70	25.4	0.67 (-1.43-2.79)	0.52	8.37	1.68 (-0.93-4.27)	0.20	4.69	0.25 (-1.63-2.12)	0.79
Non IDPN	57.6			22.7			24.8			6.69			4.44		

Month 1	Laboratory		
	Albumin		
	Mean	MD (95%CI)	<i>p</i> value
Unadjusted [‡]			
Oral	3.31	0.16 (0.01-0.31)	0.03*
Non Oral	3.47		
IDPN	3.50	0.13 (0.01-0.25)	0.04*
Non IDPN	3.37		
Adjusted [§]			
Oral	3.30	0.17 (0.03-0.32)	0.02*
Non Oral	3.47		
IDPN	3.50	0.13 (0.04-0.26)	0.04*
Non IDPN	3.37		

[†]Bivariate analysis[‡]Independent sample t test[§]ANCOVA from multiple linear regression**p*<0.05 (Independent sample t test and multiple linear regression conducted using age and duration of haemodialysis as independent variables).***p*<0.05 (Multiple linear regression conducted using age and duration of haemodialysis as confounding variables)

Table 3. Outcomes evaluated at months 1 and 3 after nutritional interventions[†] (cont.)

Month 3	Anthropometric Profile														
	Weight (kg)			Body mass index			Upper arm circumference			Triceps skinfold thickness			Biceps skinfold thickness		
	Mean	MD (95%CI)	<i>p</i> value	Mean	MD (95%CI)	<i>p</i> value	Mean	MD (95%CI)	<i>p</i> value	Mean	MD (95%CI)	<i>p</i> value	Mean	MD (95%CI)	<i>p</i> value
Unadjusted [‡]															
Oral	54.9	4.94 (-10.4-0.46)	0.72	20.9	2.82 (0.54-5.09)	0.02*	26.1	0.87 (-1.29-3.03)	0.42	11.1	4.36 (1.06-7.66)	0.01*	7.21	3.26 (0.91-5.60)	0.01*
Non Oral	59.8			23.7			25.2			6.69			3.95		
IDPN	60.0	2.70 (-3.04-8.44)	0.35	23.4	0.77 (-1.67-3.20)	0.53	25.4	0.16 (-2.14-1.80)	0.86	8.69	1.24 (-1.53-4.01)	0.38	4.64	0.45 (-2.17-1.27)	0.61
Non IDPN	57.3			22.6			25.5			7.45			5.09		
Adjusted [§]															
Oral	54.9	4.95 (-11.1-1.20)	0.11	20.9	2.82 (0.27-5.38)	0.03**	25.6	0.17 (-2.14-2.49)	0.88	10.6	3.71 (0.63-6.78)	0.02*	6.81	2.70 (0.791-4.59)	0.01*
Non Oral	59.8			23.7			25.4			6.88			4.11		
IDPN	60.0	2.20 (-8.0-3.69)	0.46	23.2	0.57 (-1.95-3.10)	0.65	25.6	0.21 (-1.81-2.24)	0.83	9.02	1.80 (-0.98-4.59)	0.20	4.89	0.01 (-1.78-1.77)	0.99
Non IDPN	57.3			22.7			25.4			7.22			4.90		

Month 3	Laboratory		
	Albumin		
	Mean	MD (95%CI)	<i>p</i> value
Unadjusted [‡]			
Oral	3.37	0.17 (0.02-0.33)	0.03*
Non Oral	3.54		
IDPN	3.56	0.12 (-0.03-0.27)	0.11
Non IDPN	3.44		
Adjusted [§]			
Oral	3.38	0.16 (0.05-0.32)	0.04*
Non Oral	3.54		
IDPN	3.56	0.11 (-0.04-0.26)	0.14
Non IDPN	3.45		

[†]Bivariate analysis

[‡]Independent sample t test

[§]ANCOVA from multiple linear regression

**p*<0.05 (Independent sample t test and multiple linear regression conducted using age and duration of haemodialysis as independent variables).

***p*<0.05 (Multiple linear regression conducted using age and duration of haemodialysis as confounding variables)

The average dry body weight of the patients in the IDPN group increased from 61.7 ± 7.7 to 63.9 ± 8.9 kg ($p=0.03$) during the intervention period, whereas that of the patients not receiving IDPN intervention remained stable (66.3 ± 10.5 to 66.3 ± 10.6 kg; $p>0.05$).¹⁶ This indicates that IDPN benefits patients' anthropometric profiles.¹⁷

Several notable results were obtained for IDPN, oral, and counselling nutrition interventions. IDPN did not appear to improve the patients' quality of life or nutrition more effectively than did oral supplementation. In the French Intradialytic Nutrition Evaluation Study (FineS), which is a randomised controlled trial (RCT) of 186 HD patients with chronic malnutrition, 1 year of IDPN administration did not worsen the mortality or hospitalisation rate or reduce quality of life. Additionally, in two RCTs, no differences were observed in BMI, serum albumin, serum prealbumin, and SGA scores between patients who received IDPN versus those who received oral supplementation. The results of these trials were limited by small sample size, nonadherence (19%–26% and 24% of patients discontinued oral supplementations and IDPN, respectively), and differences in numbers of participants between groups (17% control vs. 0% IDPN). A significant improvement in nutritional indicators was observed only in a small prospective cohort ($n=20$) from Turkey; patients receiving IDPN exhibited a more significant increase in serum albumin after 4 months than did patients who received oral supplements. However, that study did not directly compare the intervention and control groups and was limited by the lack of adherence (40% of the patients switched from oral supplements to IDPN due to nonadherence) and no statistical adjustment for confounding variables.^{18,19}

In another RCT, 107 CHD patients treated with IDPN were compared with patients treated with "regular food behaviour" counselling for 16 weeks. All the patients received nutritional counselling at baseline. In this study, IDPN did not consistently improve the patients' health or nutrition. At 4 weeks, the patients receiving IDPN exhibited serum prealbumin levels that were 15% higher than those of the patients in the control group (41% IDPN vs. 20.5% controls, $p=0.042$). However, despite this 15% difference, the difference in clinical outcomes between the groups was nonsignificant. The increase in mean serum prealbumin (26.31 mg/L) at 16 weeks did not reach the threshold of >30 mg/L. It also concluded no improvement in mortality (26.4% vs. 12.9%, $p=0.09$), hospitalisation (hospitalisation rate: 59% vs. 43.2%, $p=0.15$), or quality of life (change in SF-12 score: -2.74 vs. 0.34 , $p=1.118$). That study was limited by its small sample size, indirect results, and lack of information on the types of interventions and cointerventions potentially received by the control group.^{18,19}

In our study, differences in albumin, biceps and triceps skinfold thickness, and BMI were identified between the oral and nonoral nutrition intervention groups at months 1 and 3 after the intervention. IDPN significantly improved albumin in the first month of the intervention, but the albumin level remained steady after month 3. In addition, age and duration of HD were determined to possibly affect albumin and biceps and triceps skinfold thickness;

however, they exhibited no correlation with BMI at months 1 and 3. We assumed that the effects of age and duration of HD were mediated by acute conditions and comorbidities experienced by the patients. Unfortunately, in this study, we homogenised the comorbidity factor; therefore, we were unable to determine the effect of the patients' comorbidities on their serum albumin levels and anthropometric profiles.

IDPN generally reduces the risk of mortality and results more favourable nutritional outcomes in patients receiving IDPN than in patients with CKD receiving standard care. The largest nonrandomised study conducted to date reported that the effect of IDPN on 1-year mortality depended on serum albumin at baseline.²⁰ Patients with low baseline serum albumin (≤ 3.3 g/dL) who received IDPN exhibited a lower mortality rate than did those who did not receive IDPN (OR: 0.61–0.72; $p<0.01$). By contrast, patients with high baseline serum albumin (>3.3 g/dL) who received IDPN exhibited a similar or higher risk of mortality than those who did not receive IDPN (OR: 0.85; $p=0.10$ – 2.6 ; $p<0.005$). A smaller nonrandomised study ($n=81$) involving patients with baseline serum albumin of 3.02 g/dL also reported that patients who received IDPN exhibited higher chances of survival. Moreover, a single RCT involving 40 CHD patients with refractory anaemia reported no difference in nutrition-related functional capacity between patients who received IDPN and those who received standard care. Although numerous studies have reported that patients who receive IDPN exhibit higher mean scores on various nutritional outcomes than do those who receive usual care, these studies have been limited by small sample sizes (all except one $n < 100$), lack of information on intervention adherence, and lack of statistical adjustment for confounding variables.¹⁸ No study has reported the proportion of patients who achieved clinically significant improvements in nutritional outcomes after receiving IDPN.

Despite the differences in the results of aforementioned studies, IDPN interventions have been commonly reported to improve patients' serum albumin levels and anthropometric profiles. This trend is consistent with the findings of the present study. Although this study included only a 3-month intervention period, the increases observed in these variables affected patient mortality as a clinical outcome. This study did not evaluate the effects of inflammatory variables or include comorbidity-based stratification that may have been related to intervention outcomes. Employing a single random sample when evaluating oral nutritional therapy is an undesirable approach due to the large number of comorbidities among the patients in the sample. Hence, a study that involves with a larger sample size, more complete measurement of variables related to inflammatory markers and mortality, and a longer follow-up period is required to fully evaluate the effect of nutritional therapy on the survival rate of HD patients with CKD and malnourishment.

Conclusion

Nutritional treatment is linked to improvements in serum albumin levels and anthropometric profiles of HD patients with CKD and malnourishment. Nevertheless, addi-

tional studies are required to examine the effect of nutritional therapy on these patients' quality of life.

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AUTHOR DISCLOSURES

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