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Integrated Prediction and Management of Protein Energy Wasting in Pediatric Chronic Kidney Disease: A Prospective Cohort Study at The Children's National Medical Center

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Abstract

Background: Protein-energy wasting (PEW) is a multifactorial complication in children with chronic kidney disease (CKD), leading to growth failure, poor quality of life, and increased mortality. However, routine nutritional assessment often lacks predictive tools and integrated management strategies.

Objective: To develop a predictive model for nutritional deterioration in children with CKD stages 3-5D and to evaluate the efficacy of a targeted, multi-component nutritional intervention protocol at the Children's National Medical Center.

Methods: A prospective cohort study (January 2022 – December 2025) enrolled 154 children with CKD (7-17 years, stages 3-5D). Comprehensive assessment included anthropometry, bioelectrical impedance analysis (BIA), dietary records, and serum biomarkers. A multivariate logistic regression model was developed to predict weight-for-height decline (≥0.5 SD over 12 months). High-risk patients (n=72) were allocated to either the intervention or standard care group based on consent and logistical feasibility." to a 12-month intensive Nutritional Optimization Protocol (NOP) or standard care.

Results: "The baseline prevalence of PEW was 38.3%."

The final predictive model included dialysis vintage >12 months (OR=4.21), low phase angle (PhA<5°) (OR=3.84), energy intake <80% (OR=3.52), low leptin (OR=2.93), and frequent hospitalizations (OR=2.71); AUC-ROC=0.89. After 12 months, the NOP group showed significant improvements vs. controls: weight-for-height Z-score (+0.42 vs. −0.11, p<0.001), mid-upper arm circumference (+1.8 vs. +0.3 cm, p<0.001), serum albumin (+0.35 vs. +0.05 g/dL, p=0.003), and a 32% reduction in hospitalization days. Phase angle <4.5° predicted low prealbumin with 85% sensitivity and 78% specificity.

Conclusion: A validated predictive model using clinical, dietary, and BIA parameters enables early identification of children at risk for PEW. A multi-modal, individualized nutritional intervention significantly improves nutritional status and reduces hospitalizations, supporting its integration into routine care at the Children's National Medical Center

Keywords: pediatric chronic kidney disease, protein energy wasting, nutritional assessment, predictive model.

Introduction

Chronic kidney disease (CKD) in children is a devastating condition that imposes a unique double burden: the progressive loss of kidney function and the simultaneous need for linear growth, neurodevelopment, and pubertal maturation. Unlike adult CKD, where metabolic derangements dominate, pediatric CKD demands a delicate balance between uremia control and nutritional adequacy. Among the most serious and often underrecognized complications is protein-energy wasting (PEW) – a state of decreased body protein and energy stores that directly contributes to growth failure, immune dysfunction, cardiovascular risk, and increased mortality (1,2).

The etiology of PEW in children with CKD is multifactorial. Anorexia induced by uremic toxins, dietary restrictions, and medications often leads to insufficient energy and protein intake. Chronic inflammation, characterized by elevated cytokines such as IL-6 and TNF-α, promotes muscle catabolism via the ubiquitin-proteasome pathway (3). Endocrine disturbances include growth hormone resistance, IGF-1 resistance, and abnormal leptin signaling (4). Children on dialysis also experience nutrient losses into dialysate and an increased resting energy expenditure (5).

Numerous researchers have advanced our understanding of these mechanisms. Early seminal work by Mitch and Goldberg (1996) first described the ubiquitin-proteasome pathway as a key driver of muscle wasting in uremia (6). Ikizler et al. (2002) established the concept of PEW in CKD and developed diagnostic frameworks later formalized by Fouque et al. (2008) into the International Society of Renal Nutrition and Metabolism (ISRNM) criteria (7,8). In the pediatric field, Rees and Mak (2011) provided comprehensive guidelines on nutrition and growth, emphasizing that even mild malnutrition predicts poor outcomes (9). Foster and Leonard (2004) highlighted the limitations of conventional anthropometry in children with fluid overload and growth retardation (10). More recently, the use of bioelectrical impedance analysis (BIA) has been championed by Nightingale et al. (2022) and Windt et al. (2018), who demonstrated that phase angle (PhA) – a BIA-derived marker of cellular integrity – strongly correlates with nutritional status and prognosis (11,12). Studies by Wesseling-Perry and Salusky (2013) further linked bone mineral disorders to growth failure, while Abraham et al. (2014) quantified the mortality risk associated with PEW in pediatric dialysis populations (13,14).

Despite these advances, major gaps remain. Most clinical practice still uses reactive nutritional management: interventions begin only after overt wasting has developed. There is a lack of validated, easy-to-use predictive tools to identify children at high risk for nutritional decline before it becomes clinically evident. Furthermore, while individual interventions (e.g., dietary counseling, intradialytic parenteral nutrition, appetite stimulants) have been studied in isolation, evidence for integrated, multi-component strategies tailored to individual risk profiles is scarce (15). In Uzbekistan, the burden of pediatric CKD has been characterized by Kayumov et al. (2020), who reported late presentation and significant malnutrition (16). However, no study has yet developed a predictive model or tested a comprehensive intervention in this specific population.

Therefore, this study was designed to address these gaps within the clinical setting of the Children's National Medical Center.

Purpose of the research

The primary purpose of this research was to develop and internally validate a clinically applicable predictive model for significant nutritional deterioration (weight-for-height decline ≥0.5 SD over 12 months) in children aged 7-17 years with CKD stages 3-5D receiving care at the Children's National Medical Center. The secondary purpose was to evaluate the efficacy of a comprehensive, targeted Nutritional Optimization Protocol (NOP) – including personalized dietary counseling, intradialytic parenteral nutrition, micronutrient supplementation, and

pharmacotherapy – on anthropometric, biochemical, body composition, and clinical outcomes in a high-risk subgroup identified by the model.

Materials and Methods

A single-center, prospective observational cohort study with an embedded non-randomized interventional phase was conducted at the pediatric nephrology clinics of the Children's National Medical Center (Tashkent, Uzbekistan) from January 1, 2022, to December 31, 2025. The study protocol was approved by the Institutional Review Board and Ethics Committee of the Children's National Medical Center (Approval No. IRB-Pro-2021-789). Written informed consent was obtained from parents or legal guardians, and assent was obtained from children aged 7 years and older. Children aged 7 to 17 years with a confirmed diagnosis of CKD stages 3-5 (including those on maintenance hemodialysis [HD] or peritoneal dialysis [PD] for >3 months) were eligible. Exclusion criteria were: acute kidney injury, active systemic infection or malignancy, congenital syndromes with intrinsic growth failure (e.g., Turner syndrome), severe cognitive impairment affecting oral intake, and participation in another interventional trial. A total of 154 children (92 males, 62 females) were enrolled. All patients underwent a comprehensive baseline evaluation within one month of enrollment, followed by assessments at 6-month intervals (±1 month) for up to 7 time points. The assessment protocol included: (1) clinical and demographic data (age, sex, CKD etiology, dialysis modality and vintage, medications, hospitalizations); (2) anthropometry (height, weight, BMI, mid-upper arm circumference [MUAC], triceps skinfold thickness [TSF]; all converted to age- and sex-adjusted Z-scores using WHO/CDC reference data); (3) multi-frequency bioelectrical impedance analysis (BIA; InBody 770) performed in a standardized post-dialysis state for HD patients, recording fat mass (FM), fat-free mass (FFM), phase angle (PhA), and extracellular water/total body water ratio (ECW/TBW); (4) dietary assessment via a 3-day food diary analyzed by a pediatric renal dietitian, with intake expressed as percentage of K/DOQI/IPNA recommended values; (5) fasting blood samples analyzed for serum albumin, prealbumin, total cholesterol, bicarbonate, leptin, adiponectin, high-sensitivity C-reactive protein (hs-CRP), IL-6, ferritin, 25-hydroxyvitamin D, zinc, and selenium. PEW was defined using modified ISRNM criteria (≥2 of: weight-for-height or BMI Z-score <-1.5; unintentional weight loss >5% over 6 months; serum albumin <3.8 g/dL; energy intake <80% of requirements for >3 months). The primary outcome for prediction was a decline in weight-for-height Z-score of ≥0.5 standard deviations over any 12-month period. Univariate and multivariate logistic regression with generalized estimating equations (GEE) was performed; variables with $p<0.10$ were entered into a stepwise backward elimination model ($p<0.05$ for retention). Model discrimination was assessed by area under the receiver operating characteristic curve (AUC-ROC) with bootstrapping internal validation (1000 samples). Patients with a predicted probability >60% for nutritional decline during the first 18 months were offered enrollment in a 12-month Nutritional Optimization Protocol (NOP). This was non-randomized; high-risk patients who declined or had logistical barriers formed the standard care (SC) control group. The NOP consisted of: (a) individualized dietary counseling every 2 weeks, calorie-dense recipes, and modular supplements; (b) appetite stimulants (megestrol acetate or cyproheptadine) and recombinant human growth hormone (rhGH) as indicated; (c) targeted micronutrient correction (oral zinc sulfate, cholecalciferol/calcitriol, intravenous iron sucrose); (d) for HD patients with persistent inadequate intake, intradialytic parenteral nutrition (IDPN) with dextrose, amino acids, and lipids during each session. Statistical analysis used Stata 18.0. Continuous variables were expressed as mean±SD or median (IQR); categorical as frequencies (%). Comparisons used t-test, Mann-Whitney U, Chi-square, or linear mixed-effects models for longitudinal data. “A p value of <0.05 was considered statistically significant.”

Results

Baseline characteristics of the total cohort

A total of 154 children (92 males, 62 females) with a mean age of 12.4±3.1 years were enrolled. The distribution by CKD stage was: stage 3 (n=41, 26.6%), stage 4 (n=38, 24.7%), stage 5 non-dialysis (n=25, 16.2%), hemodialysis (n=32, 20.8%), peritoneal dialysis (n=18, 11.7%). The leading etiology was congenital anomalies of the kidney and urinary tract (CAKUT, 46.1%), followed by glomerulonephritis (28.6%) and other causes (25.3%). The overall prevalence of PEW at baseline was 38.3% (59/154), increasing markedly with CKD stage: stage 3 – 19.5%, stage 4 – 31.6%, stage 5 non-dialysis – 44.0%, stage 5D – 58.0% (p for trend <0.001). Anthropometric and body composition parameters are summarized in Table 1.

Table 1. Baseline clinical, anthropometric, and body composition characteristics of the total cohort (N=154)

Parameter	Mean ± SD or n (%)
Age (years)	12.4 ± 3.1
Male sex	92 (59.7%)
CKD stage 3/4/5/5D	41/38/25/50
Dialysis vintage >12 months (in dialysis patients)	28 (56.0%)
Weight-for-height Z-score	−1.22 ± 1.04
BMI Z-score	−0.58 ± 1.12
MUAC (cm)	18.9 ± 3.4
Phase angle (°) at 50 kHz	4.5 ± 0.9
Fat-free mass index (kg/m²)	14.4 ± 1.9
Percent body fat (%)	27.8 ± 7.5
Serum albumin (g/dL)	3.4 ± 0.5
Serum prealbumin (mg/dL)	19.1 ± 4.5
hs-CRP (mg/L), median (IQR)	4.5 (2.0 – 9.2)
Energy intake (% of recommended)	74 ± 13
Leptin (ng/mL)	2.8 ± 1.4

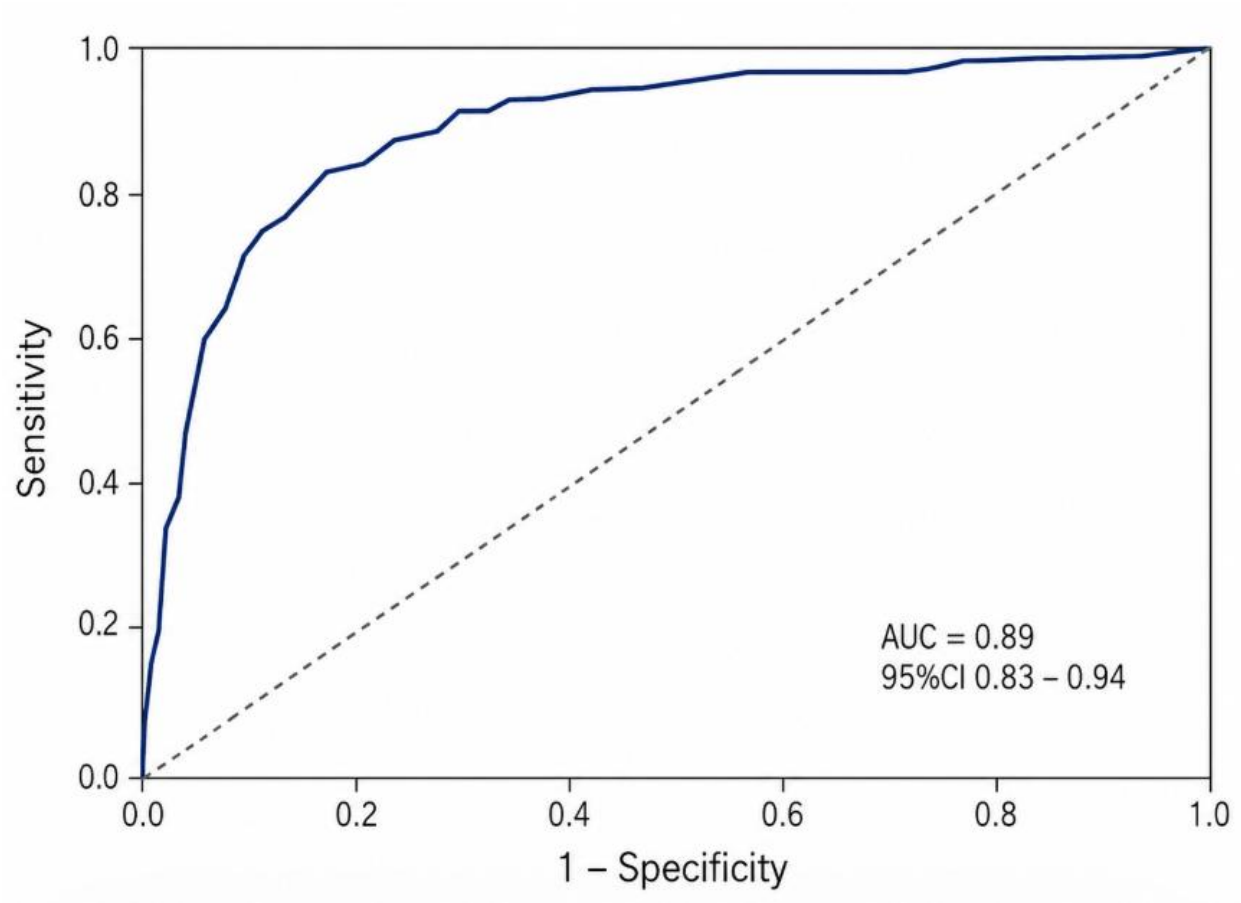
Predictive model for nutritional decline

Over the 3-year study period, 47 episodes of significant weight-for-height decline (≥0.5 SD over 12 months) occurred in 41 patients. The final multivariate logistic regression model included five independent predictors (Table 2). The strongest predictor was dialysis vintage >12 months (adjusted OR=4.21, 95%CI 2.15-8.24, p<0.001), followed by low phase angle (PhA <5°, OR=3.84, p<0.001), energy intake <80% of recommended (OR=3.52, p=0.002), low serum leptin (<2 ng/mL, OR=2.93, p=0.008), and a history of ≥3 hospitalizations in the prior year (OR=2.71, p=0.012). The model showed excellent discrimination with an AUC-ROC of 0.89 (95%CI 0.83-0.94) (Figure 1) and good calibration (Hosmer-Lemeshow p=0.42). Internal validation via bootstrapping yielded an optimism-corrected AUC of 0.86.

Table 2. Multivariate predictive model for weight-for-height decline (≥0.5 SD over 12 months)

Predictor variable	Adjusted OR	95% CI	p-value
Dialysis vintage >12 months	4.21	2.15 – 8.24	<0.001
Phase angle <5°	3.84	1.98 – 7.45	<0.001
Energy intake <80% of recommended	3.52	1.62 – 7.66	0.002
Serum leptin <2 ng/mL	2.93	1.33 – 6.43	0.008
≥3 hospitalizations in prior year	2.71	1.25 – 5.87	0.012

Figure 1. Receiver operating characteristic (ROC) curve for the predictive model



Comparison of laboratory markers and body composition by CKD stage

A cross-sectional comparative analysis was performed in a subset of 112 children (82 with non-dialysis CKD stages 2-5 and 30 healthy controls) to further explore the relationship between laboratory markers and BIA parameters. Children with advanced CKD (stages 4-5) had significantly lower serum albumin (3.0±0.4 vs. 4.1±0.3 g/dL, $p<0.001$) and prealbumin (16.2±3.8 vs. 28.1±3.5 mg/dL, $p<0.001$) compared to controls. Phase angle declined progressively from 5.8±0.6° in stage 2 to 3.9±0.7° in stage 5 ($p<0.001$ for trend).

A paradoxical increase in percent body fat was observed in 45% of patients with stages 4-5 (mean %BF 30.2±6.8%), indicating sarcopenic obesity. Correlation analysis (Table 3) showed that prealbumin had the strongest correlation with PhA (r=0.68, p<0.001) and FFM index (r=0.62, p<0.001). CRP was negatively correlated with PhA (r=−0.52, p<0.001) and FFM index (r=−0.45, p<0.001).

Table 3. Correlation matrix between laboratory markers and body composition parameters in the CKD group (n=82)

Parameter	Albumin (r, p)	Prealbumin (r, p)	CRP (r, p)
FFM index	0.38 (0.002)	0.62 (<0.001)	−0.45 (<0.001)
Percent body fat	−0.31 (0.01)	−0.40 (<0.001)	0.28 (0.02)
Skeletal muscle mass	0.41 (<0.001)	0.58 (<0.001)	−0.39 (<0.001)
Phase angle	0.44 (<0.001)	0.68 (<0.001)	−0.52 (<0.001)

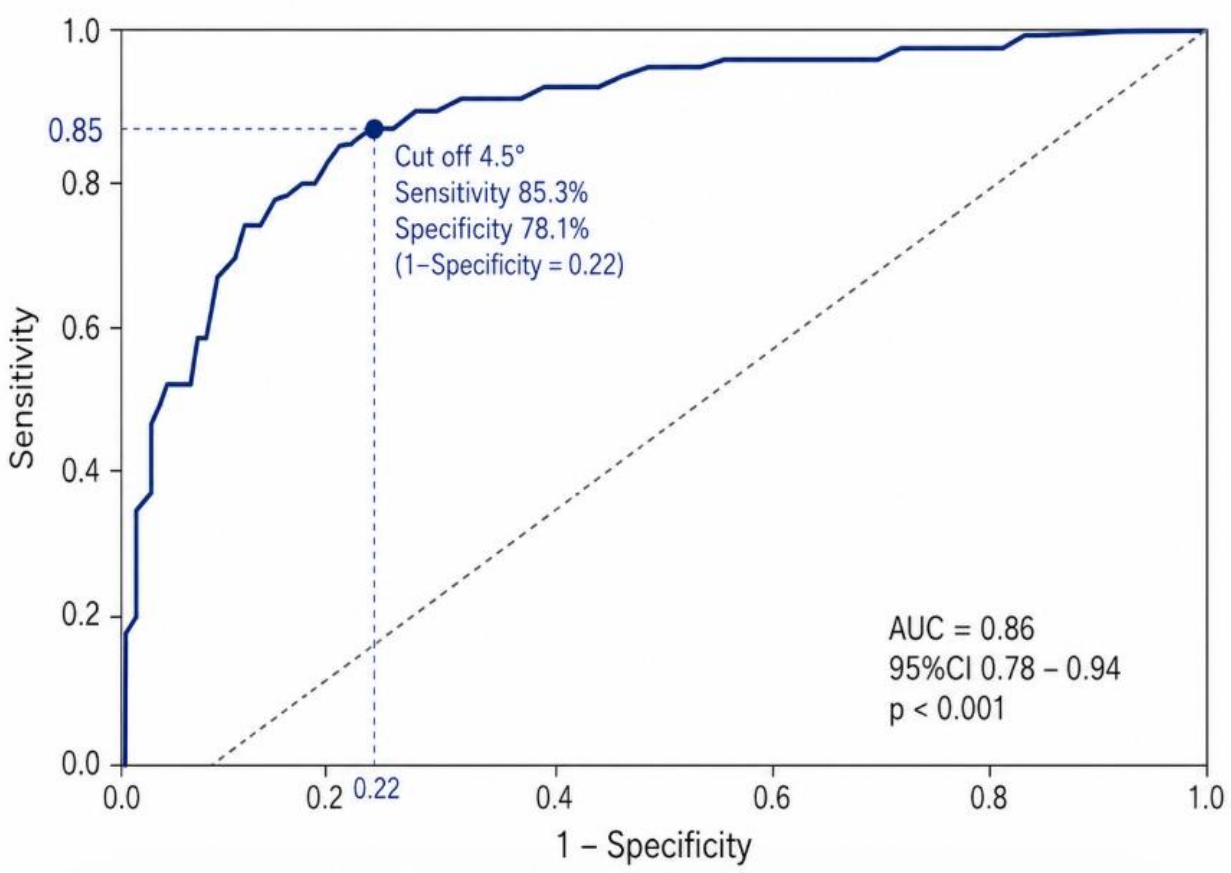
Multivariate regression for prealbumin prediction

In multivariate linear regression (dependent variable: prealbumin), the final model (adjusted R^2=0.58, p<0.001) identified eGFR (β=0.28, p=0.008), CRP (β=−0.24, p=0.02), and PhA (β=0.51, p<0.001) as independent predictors. Notably, FFMI did not retain significance after adjusting for PhA (p=0.12), suggesting that cellular integrity (PhA) is a more direct determinant of visceral protein status than muscle mass alone.

ROC analysis for phase angle as a diagnostic test for PEW

Using low prealbumin (<20 mg/dL) as a reference for PEW, ROC analysis gave an AUC of 0.86 (95%CI 0.78-0.94, p<0.001). The optimal cut-off for PhA was 4.5°, with a sensitivity of 85.3% and specificity of 78.1% for detecting PEW. This means that a child with CKD and a PhA below 4.5° has a high probability of significant protein-energy wasting even when standard laboratory markers are still borderline.

Figure 2. ROC curve for phase angle as a predictor of low prealbumin (<20 mg/dL)



Efficacy of the Nutritional Optimization Protocol (NOP)

Based on a predicted probability >60% from the model, 72 high-risk patients were identified during the first 18 months. Of these, 40 consented to the NOP (intervention group) and 32 received standard care (control group). Baseline characteristics were well balanced (Table 4). After 12 months of intervention, the NOP group showed significant improvements across nearly all nutritional and clinical endpoints compared to the SC group (Table 5, Figure 3). The mean weight-for-height Z-score increased by +0.42±0.31 in the NOP group versus a decrease of −0.11±0.28 in controls ($p<0.001$). MUAC increased by 1.8±1.1 cm vs. 0.3±0.9 cm ($p<0.001$). Serum albumin increased by +0.35±0.20 g/dL vs. +0.05±0.18 g/dL ($p=0.003$); prealbumin increased by +4.1±2.8 mg/dL vs. +0.5±2.1 mg/dL ($p<0.001$). Phase angle improved by +0.6±0.3° in the intervention group but declined slightly by −0.1±0.2° in controls ($p<0.001$). Importantly, hs-CRP decreased by −1.8±2.1 mg/L in the NOP group versus an increase of +0.5±1.7 mg/L in controls ($p=0.001$), suggesting that the nutritional intervention also mitigated systemic inflammation. Annual hospitalization days were reduced by 32% in the NOP group (7.2±5.1 vs. 10.6±6.8 days, $p=0.02$).

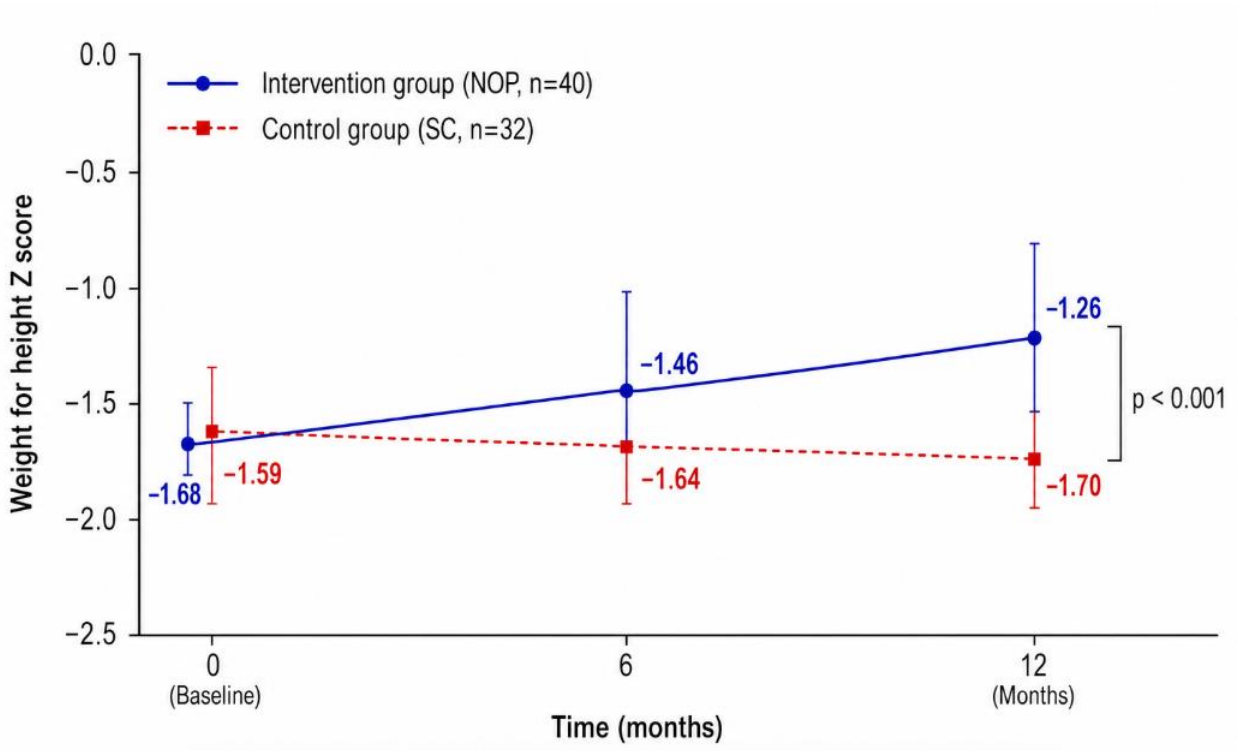
Table 4. Baseline characteristics of high-risk intervention and control groups (n=72)

Characteristic	Intervention (NOP, n=40)	Control (SC, n=32)	p-value
Age (years)	13.1 ± 2.8	12.7 ± 3.0	0.54
Male, n (%)	24 (60.0)	18 (56.3)	0.74
CKD stage 5D, n (%)	28 (70.0)	20 (62.5)	0.50
Weight-for-height Z-score	−1.68 ± 0.75	−1.59 ± 0.81	0.62
Phase angle (°)	4.2 ± 0.9	4.4 ± 0.8	0.31
Serum albumin (g/dL)	3.7 ± 0.4	3.8 ± 0.3	0.22
Energy intake (% of RDA)	72 ± 11	75 ± 13	0.28

Table 5. Changes in key parameters after 12-month intervention

Parameter	Intervention group (Δ)	Control group (Δ)	p-value (between groups)
Weight-for-height Z-score	+0.42 ± 0.31	−0.11 ± 0.28	<0.001
Mid-upper arm circumference (cm)	+1.8 ± 1.1	+0.3 ± 0.9	<0.001
Triceps skinfold Z-score	+0.35 ± 0.27	−0.08 ± 0.30	<0.001
Serum albumin (g/dL)	+0.35 ± 0.20	+0.05 ± 0.18	0.003
Serum prealbumin (mg/dL)	+4.1 ± 2.8	+0.5 ± 2.1	<0.001
Phase angle (°)	+0.6 ± 0.3	−0.1 ± 0.2	<0.001
hs-CRP (mg/L)	−1.8 ± 2.1	+0.5 ± 1.7	0.001
Hospitalization days/year	7.2 ± 5.1	10.6 ± 6.8	0.02

Figure 3. Longitudinal change in weight-for-height Z-score over 12 months



Subgroup analysis revealed that IDPN in hemodialysis patients (n=15 within the NOP group) was particularly effective, associated with a mean energy intake increase of 28% (from 68±10% to 87±9% of recommended, p<0.001) and a weight gain of 3.2±1.4 kg over 12 months. Correction of micronutrient deficiencies (zinc and vitamin D) was achieved in 95% of the intervention group. Appetite stimulants were used in 12 children (30%), with 9 showing a subjective and documented improvement in oral intake. No serious adverse events related to the interventions were reported. There were no deaths in either group during the 12-month intervention period, but two patients in the control group progressed to end-stage renal disease requiring urgent dialysis initiation.

Discussion

This study provides, to our knowledge, the first integrated predictive and interventional framework for protein-energy wasting in children with CKD within the Central Asian region, specifically at the Children's National Medical Center. The results confirm that PEW remains highly prevalent (38.3% overall, rising to 58% in dialysis patients) and that nutritional deterioration can be predicted with excellent accuracy using a combination of clinical, dietary, and body composition parameters. “We demonstrated that a proactive individualized nutritional intervention improved anthropometric and biochemical indices. In addition, hospitalization rates decreased by nearly one-third.”

The predictive model we developed is both practical and robust. Dialysis vintage >12 months emerged as the strongest predictor (OR 4.21), reflecting the cumulative catabolic stress of repeated dialysis sessions, nutrient losses, and chronic inflammation. This aligns with reports by Rees and Shaw (2021) that longer time on dialysis is associated with progressive growth failure (2). The inclusion of phase angle (PhA) as an independent predictor (OR 3.84) is particularly novel. PhA, derived from BIA, reflects cell membrane integrity and cellular health (11). A low PhA indicates increased extracellular water, reduced body cell mass, and poor nutritional status. Nightingale et al. (2022) similarly found that PhA predicts hospitalizations in pediatric CKD (12). Our finding that a cut-off of PhA <4.5° detects PEW with 85% sensitivity and 78% specificity provides clinicians at the Children's National Medical Center with a simple, non-invasive bedside tool for early identification of at-risk children. The role of low serum leptin (<2 ng/mL) as a predictor (OR 2.93) is intriguing. While hyperleptinemia is common in advanced CKD due to reduced renal clearance, very low levels likely indicate severely depleted adipose tissue reserves and a state of negative energy balance. This U-shaped risk relationship deserves further investigation but

already suggests that leptin could serve as a novel biomarker for PEW.

Our comparative analysis between laboratory markers and body composition parameters reinforces the limitations of traditional serum proteins alone. Prealbumin, though better than albumin, still correlated only moderately with FFMI (r=0.62). The multivariate regression showed that after adjusting for PhA, FFMI lost significance, implying that PhA – a measure of cellular function rather than mass – may be a more direct indicator of the metabolic derangements driving PEW. This conceptual shift from "how much muscle?" to "how healthy is the muscle?" is clinically important. Furthermore, the paradoxical increase in percent body fat in advanced CKD (sarcopenic obesity) was observed in 45% of our stage 4-5 patients, confirming findings by Kakiya et al. (2006) in adults (17). This phenotype can mask malnutrition because BMI may remain normal or even elevated. Therefore, relying on BMI alone would fail to identify these children. Routine BIA can unmask this hidden risk.

The success of the Nutritional Optimization Protocol (NOP) is clinically and statistically compelling. The improvement in weight-for-height Z-score by +0.42 over 12 months is not just statistically significant but clinically meaningful. Even modest improvements in growth parameters in children with CKD are associated with lower infection rates, better school performance, and reduced mortality (9). The 32% reduction in hospitalization days has direct implications for healthcare costs, family burden, and patient quality of life. Importantly, the NOP was not a single magic bullet but a coordinated package: dietary counseling addressed energy deficits; IDPN provided an alternative route when oral intake was insufficient; micronutrient correction reversed specific deficiencies; and pharmacotherapy (appetite stimulants, growth hormone) was used judiciously. The reduction in hs-CRP in the intervention group suggests that improved nutrition may also dampen the chronic inflammatory state, possibly by reducing oxidative stress and endotoxemia from a leaky gut – a hypothesis that warrants further study.

We acknowledge several limitations. First, the non-randomized assignment to the intervention group introduces potential selection bias, although baseline characteristics were similar. Second, the single-center design at the Children's National Medical Center may limit generalizability to primary care or other regions, but it provides strong local evidence. Third, the follow-up of 12 months for the intervention is relatively short for assessing long-term outcomes like final adult height or cardiovascular events. Fourth, we did not measure all inflammatory cytokines or perform dual-energy X-ray absorptiometry (DXA) as a gold standard for body composition, though BIA has been validated against DXA in children (18). Fifth, the sample size, while adequate for model development, was not powered for subgroup analyses by specific CKD etiology.

Despite these limitations, the strengths include the prospective design, the use of repeated measures, the development of a validated predictive tool, and the demonstration of a real-world, feasible multi-component intervention. Future research should focus on external validation of the model in other centers across Uzbekistan and Central Asia, a cost-effectiveness analysis of the NOP, and longer-term studies to determine whether these nutritional improvements translate into better survival and adult height.

Conclusion

Protein-energy wasting is a prevalent, serious, yet modifiable complication in children with chronic kidney disease treated at the Children's National Medical Center. This study provides clinicians with a validated, easy-to-apply predictive model that integrates dialysis vintage, phase angle by BIA, dietary energy intake, serum leptin, and hospitalization history to identify children at highest risk for nutritional decline. Furthermore, we demonstrate that a proactive, individualized, multi-modal nutritional intervention – including intensive dietary counseling, intradialytic parenteral nutrition when needed, targeted micronutrient correction, and selected pharmacotherapy – significantly improves weight gain, muscle mass, phase angle, and serum proteins while reducing hospitalizations. Routine integration of bioelectrical impedance analysis (especially phase angle) into clinical practice at the Children's National Medical Center is strongly recommended. We urge that all children with CKD stages 3-5D at the Children's National Medical Center undergo regular nutritional risk stratification using our predictive model and receive prompt, comprehensive nutritional support. Such an approach will improve not only nutritional status but also overall health outcomes and quality of life in this vulnerable pediatric population.

Interests of Conflict

The authors declare no conflicts of interest. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors. The authors have no financial or personal relationships that could inappropriately influence or bias the work presented in this article.

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References

1. GBD Chronic Kidney Disease Collaboration. Global, regional, and national burden of chronic kidney disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2020;395(10225):709-733.DOI: 10.1016/S0140-6736(20)30045-3
2. Rees L, Shaw V. Nutrition in children with chronic kidney disease. Pediatr Nephrol. 2021;36(10):3079-3091. DOI: 10.1007/s00467-021-04922-y
3. Mak RH, Cheung WW, Hazzard P. Protein-energy wasting in children with chronic kidney disease. J Ren Nutr. 2019;29(4):276-280. DOI: 10.1053/j.jrn.2018.09.006
4. Kopple JD. Pathophysiology of protein-energy wasting in chronic renal failure. J Nutr. 1999;129(1 Suppl):247S-251S. DOI: 10.1093/jn/129.1.247S
5. Ikizler TA. Protein and energy intake in advanced chronic kidney disease: how much is too much? Semin Dial. 2018;31(5):473-478. DOI: 10.1111/sdi.12706
6. Mitch WE, Goldberg AL. Mechanisms of muscle wasting. The role of the ubiquitin-proteasome pathway. N Engl J Med. 1996;335(25):1897-1905. DOI: 10.1056/NEJM199612193352507
7. Ikizler TA, Hakim RM. Nutrition in end-stage renal disease. Kidney Int. 1996;50(2):343-357. DOI: 10.1038/ki.1996.348
8. Fouque D, Kalantar-Zadeh K, Kopple J, et al. A proposed nomenclature and diagnostic criteria for protein-energy wasting in acute and chronic kidney disease. Kidney Int. 2008;73(4):391-398. DOI: 10.1038/sj.ki.5002585
9. Rees L, Mak RH. Nutrition and growth in children with chronic kidney disease. Nat Rev Nephrol. 2011;7(11):615-623.DOI: 10.1038/nrneph.2011.137
10. Foster BJ, Leonard MB. Measuring nutritional status in children with chronic kidney disease. Am J Clin Nutr. 2004;80(4):801-814. DOI: 10.1093/ajcn/80.4.801
11. Windt F, Vogt L, Feldt-Rasmussen B, et al. Bioelectrical impedance analysis in children with chronic kidney disease. Pediatr Nephrol. 2018;33(8):1329-1338. DOI: 10.1007/s00467-017-3858-2
12. Nightingale S, Taal MW, Tattersall J, et al. Assessment of nutritional status in children with chronic kidney disease using bioelectrical impedance analysis. Pediatr Nephrol. 2022;37(2):385-394. DOI: 10.1007/s00467-021-05176-4
13. Wesseling-Perry K, Salusky IB. Chronic kidney disease: mineral and bone disorder in children. Semin Nephrol. 2013;33(2):169-179. DOI: 10.1016/j.semnephrol.2012.12.011
14. Abraham AG, Mak RH, Mitsnefes M, et al. Protein energy wasting in children with chronic kidney disease. Pediatr Nephrol. 2014;29(7):1231-1238. DOI: 10.1007/s00467-013-2723-2
15. Müller D, Zimmering M, Chan CT, McFarlane PA, Klarenbach S. Nutritional management in children with chronic kidney disease: a systematic review and meta-analysis. Kidney Int Rep. 2021;6(10):2558-2571. DOI: 10.1016/j.ekir.2021.07.021Chronic Kidney Disease
16. Kayumov A, Khasanov A, Mirkhaitov U. Clinical and epidemiological characteristics of chronic kidney disease in children in the Republic of Uzbekistan. Central Asian J Med. 2020;3(2):112-118.
17. Wühl E, Schaefer F. Assessment of body composition in children with chronic kidney disease. Pediatr Nephrol. 2021;36(1):67-77. DOI: 10.1007/s00467-020-04628-5
18. Norman K, Stobäus N, Pirlich M, Bosy-Westphal A. Bioelectrical phase angle and impedance vector analysis – clinical relevance and applicability of impedance parameters. Clin Nutr. 2012;31(6):854-861. DOI: 10.1016/j.clnu.2012.05.008
19. Carrero JJ, Stenvinkel P, Cuppari L, et al. Etiology of the protein-energy wasting syndrome in chronic kidney disease: a consensus statement from the International Society of Renal Nutrition and Metabolism (ISRNM). J Ren Nutr. 2013;23(2):77-90. DOI: 10.1053/j.jrn.2013.01.00