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NUTRITIONAL STATUS AND GROWTH PATTERNS IN CHILDREN WITH CHRONIC KIDNEY DISEASE: A HOSPITAL-BASED CROSS-SECTIONAL ANALYSIS

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Abstract

Introduction: Chronic kidney disease (CKD) in children is associated with significant growth failure and malnutrition, with limited data available from developing countries. This study aimed to assess nutritional status and growth patterns in pediatric CKD patients and evaluate relationships between nutritional parameters and growth outcomes.

Methods: A hospital-based cross-sectional study was conducted at People's College of Medical Sciences & Research Centre, Bhopal, from July to December 2013. One hundred children aged 2-18 years with CKD stages 2-5 (not on dialysis) were enrolled using consecutive sampling. Comprehensive assessment included anthropometric measurements, biochemical analysis, and dietary evaluation using 24-hour recall and food frequency questionnaires. Growth parameters were expressed as Z-scores using WHO standards.

Results: The study population had a mean age of 9.2±4.1 years with 62% males. Growth failure was highly prevalent with 69% having below-normal height-for-age and 45% showing stunting. Malnutrition affected 66% of children, increasing from 44.4% in Stage 2 to 75.0% in Stage 5 CKD. Only 34% met recommended caloric intake and 42% met protein requirements. Strong positive correlations were observed between serum albumin (r=0.64), hemoglobin (r=0.56), energy intake (r=0.48), and height Z-scores. Anemia was universal, and secondary hyperparathyroidism affected all advanced CKD stages. Early disease onset and longer CKD duration were significantly associated with worse growth outcomes.

Conclusion: Children with CKD demonstrate high prevalence of growth failure and malnutrition that worsen with disease progression. Comprehensive nutritional intervention beginning in early CKD stages, addressing dietary inadequacy, anemia, and mineral metabolism disorders, is essential for optimizing growth outcomes in this vulnerable population.

Keywords: chronic kidney disease, pediatric nutrition, growth failure, malnutrition, stunting

Introduction

Chronic kidney disease (CKD) in children represents a significant global health challenge with profound implications for growth, development, and long-term quality of life. The prevalence of pediatric CKD has been steadily increasing worldwide, with congenital anomalies of the kidney and

urinary tract (CAKUT) being the leading cause in children, accounting for approximately 50-60% of cases (Harambat et al., 2012). In developing countries like India, the burden of pediatric CKD presents unique challenges due to limited healthcare resources, delayed diagnosis, and complex socioeconomic factors that influence disease progression and management outcomes.

Growth failure remains one of the most devastating complications of pediatric CKD, affecting approximately 30-60% of children with advanced stages of the disease (Seikaly et al., 2006). The pathophysiology of growth retardation in CKD is multifactorial, involving complex interactions between nutritional deficiencies, metabolic acidosis, chronic inflammation, anemia, renal osteodystrophy, and disruption of the growth hormone-insulin-like growth factor-I (GH-IGF-I) axis (Mehls et al., 2008). The severity of growth impairment correlates directly with the degree of renal dysfunction, with children developing CKD at younger ages being particularly vulnerable to significant height deficits that persist into adulthood.

Malnutrition, encompassing both protein-energy wasting and micronutrient deficiencies, serves as a critical modifiable risk factor for poor growth outcomes in pediatric CKD. The uremic environment creates a complex milieu that promotes catabolism while simultaneously reducing appetite and nutrient intake through various mechanisms including chronic inflammation, metabolic acidosis, and accumulation of uremic toxins (Mak et al., 2005). Children with CKD frequently experience reduced taste sensation, early satiety, nausea, and vomiting, all of which contribute to inadequate caloric and protein intake essential for normal growth and development.

The assessment of nutritional status in children with CKD presents unique challenges due to altered body composition, fluid retention, and the absence of a single reliable marker for nutritional assessment (Foster & Leonard, 2004). Traditional anthropometric measurements require careful interpretation in the context of fluid status and growth retardation, while biochemical markers may be influenced by the underlying kidney disease and its treatment. The Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines emphasize the importance of comprehensive nutritional assessment using multiple parameters including dietary intake evaluation, anthropometric measurements, and biochemical markers to accurately characterize nutritional status in this vulnerable population (KDOQI Work Group, 2009).

International studies have consistently demonstrated the strong association between nutritional status and growth outcomes in children with CKD. The North American Pediatric Renal Transplant Cooperative Study (NAPRTCS) data revealed that approximately one-third of children with CKD achieved final heights below the third percentile, with the degree of growth impairment being most pronounced in those who developed kidney disease during infancy (Fivush et al., 1998). European studies have similarly shown that early-onset CKD results in more severe growth deficits, with the potential for some catch-up growth following successful renal replacement therapy, though complete normalization of height is rarely achieved (Haffner et al., 2000).

Research from developing countries, particularly from the Indian subcontinent, has highlighted additional challenges in managing pediatric CKD, including delayed presentation, limited access to specialized care, and socioeconomic barriers to optimal nutrition management. Studies from Indian centers have reported higher rates of malnutrition and more severe growth retardation compared to developed countries, emphasizing the need for region-specific research to understand local patterns and develop appropriate interventions (Norman et al., 2000).

The growth hormone-IGF-I axis dysfunction in pediatric CKD represents a complex interplay of uremic toxins, chronic inflammation, and nutritional deficiencies. Children with CKD typically exhibit GH resistance rather than GH deficiency, characterized by elevated GH levels but reduced IGF-I bioactivity due to increased levels of IGF-binding proteins and reduced GH receptor expression (Tönshoff et al., 1996). This pathophysiological understanding has led to the use of recombinant human growth hormone therapy as an effective intervention for improving linear growth in children with CKD, though optimal outcomes are achieved when nutritional status is simultaneously optimized.

The relationship between nutritional status and growth in pediatric CKD extends beyond simple caloric adequacy to encompass complex metabolic interactions involving protein metabolism, mineral homeostasis, acid-base balance, and chronic inflammation. Metabolic acidosis, a common complication of CKD, directly impairs growth through multiple mechanisms including increased protein catabolism, reduced protein synthesis, and interference with GH action (Kraut & Madias, 2011). Similarly, disturbances in calcium-phosphorus homeostasis and secondary hyperparathyroidism contribute to both growth impairment and bone disease through complex effects on the growth plate and skeletal development.

Contemporary understanding of the "malnutrition-inflammation-cachexia syndrome" in CKD has revealed the central role of chronic inflammation in perpetuating both malnutrition and growth failure. Elevated levels of pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha not only suppress appetite and promote protein catabolism but also interfere with the anabolic actions of growth hormone and IGF-I (Mak et al., 2006). This inflammatory state, combined with the uremic environment, creates a vicious cycle where malnutrition promotes inflammation, which in turn worsens nutritional status and growth outcomes.

The assessment of dietary intake in children with CKD requires specialized expertise due to the complex interplay between required restrictions and growth needs. While adult CKD patients often benefit from protein restriction to reduce uremic toxin accumulation, children require adequate protein intake to support growth and development, creating a delicate balance that must be individualized based on the stage of CKD, growth velocity, and nutritional status (Chaturvedi & Jones, 2007). Similarly, mineral restrictions necessary to prevent bone disease must be carefully balanced against the requirements for normal skeletal development.

Recent advances in understanding the pathophysiology of growth failure in pediatric CKD have highlighted the importance of early intervention and comprehensive management approaches. The concept of "catch-up growth" following renal replacement therapy has been challenged by long-term follow-up studies showing that significant height deficits often persist into adulthood, emphasizing the critical importance of optimizing growth during the pre-dialysis period (Fine et al., 2010). This understanding has led to more aggressive approaches to nutritional management and earlier consideration of growth hormone therapy in children with CKD-related growth failure.

The socioeconomic and cultural factors influencing nutritional status in children with CKD are particularly relevant in the Indian context, where dietary practices, food security, and healthcare access vary significantly across different regions and socioeconomic strata. Traditional dietary patterns, religious food restrictions, and economic constraints may limit the ability to provide optimal nutrition for children with CKD, necessitating culturally sensitive and economically feasible intervention strategies.

Given the complexity of factors influencing nutritional status and growth in pediatric CKD, comprehensive assessment studies are essential to understand local patterns, identify modifiable risk factors, and develop evidence-based management strategies. The relationship between various anthropometric, biochemical, and dietary parameters requires systematic evaluation to establish appropriate screening tools and intervention thresholds for use in clinical practice.

The aim of the study is to assess the nutritional status and growth patterns in children with chronic kidney disease and to evaluate the relationship between various nutritional parameters and growth outcomes in a hospital-based setting.

Methodology

Study Design

This investigation was conducted as a hospital-based cross-sectional analytical study to examine the nutritional status and growth patterns in pediatric patients with chronic kidney disease.

Study Site
The study was conducted at **People's College of Medical Sciences & Research Centre**, Bhopal, a tertiary care teaching hospital serving as a major referral center for pediatric nephrology services in Central India.

Study Duration
The study was conducted over a period of 6 months from July 2013 to December 2013.

Sampling and Sample Size
The study employed a consecutive sampling method to recruit pediatric patients with chronic kidney disease attending the outpatient department and those admitted to the pediatric ward during the study period. Consecutive sampling was chosen as the most appropriate non-probability sampling technique for this clinical setting, as it ensured the inclusion of all eligible patients presenting during the study period while minimizing selection bias. This approach was particularly suitable given the specialized nature of pediatric nephrology care and the relatively limited number of patients seen in this tertiary care setting. The sample size was determined based on the expected prevalence of malnutrition in children with CKD (approximately 30-40% based on previous studies) and the need to detect clinically significant associations between nutritional parameters and growth outcomes. Using a precision of 10% and a confidence level of 95%, a minimum sample size of 80 patients was calculated. However, to account for potential dropouts, incomplete data, and to improve the statistical power of the study, a target sample size of 100 patients was established. The final sample consisted of all eligible patients who met the inclusion criteria and provided informed consent during the study period.

Inclusion and Exclusion Criteria
Children aged 2 to 18 years with chronic kidney disease stages 2-5 (not on dialysis) as defined by the KDOQI guidelines, attending the pediatric nephrology clinic or admitted to the pediatric ward during the study period, were included in the study. Patients with stable chronic kidney disease for at least 3 months prior to enrollment were considered eligible to ensure that acute fluctuations in renal function or recent interventions would not confound the nutritional assessment. Both male and female patients were included without gender restrictions to ensure representative sampling. Exclusion criteria included children with acute kidney injury or acute-on-chronic kidney disease, patients on any form of renal replacement therapy (hemodialysis, peritoneal dialysis, or renal transplantation), children with other chronic illnesses that could independently affect nutritional status or growth (such as inflammatory bowel disease, malignancy, or endocrine disorders), patients receiving growth hormone therapy or other anabolic agents, children with severe developmental disabilities that would preclude accurate anthropometric measurements, and those whose parents or guardians did not provide informed consent for participation in the study.

Data Collection Tools and Techniques
Data collection was performed using standardized protocols and validated instruments to ensure consistency and reliability of measurements. A structured questionnaire was developed to collect demographic information, medical history, and clinical data including age, gender, primary renal diagnosis, duration of CKD, family history of kidney disease, and current medications. Anthropometric measurements were obtained using standardized techniques following WHO guidelines, with height measured using a wall-mounted stadiometer (accurate to 0.1 cm), weight measured using a calibrated digital scale (accurate to 0.1 kg), and head circumference measured using a non-stretchable measuring tape in children below 5 years of age. Mid-upper arm circumference and triceps skinfold thickness were measured using standard techniques with appropriate instruments (flexible measuring tape and Harpenden calipers, respectively) to assess muscle mass and subcutaneous fat stores. All anthropometric measurements were converted to age

and gender-specific Z-scores using WHO growth reference standards for international comparison. Dietary assessment was conducted using a 24-hour dietary recall method administered by a trained dietitian, with portion sizes estimated using standard household measures and food models. A semi-quantitative food frequency questionnaire was also administered to assess usual dietary patterns over the preceding month. Blood samples were collected after appropriate fasting (8-12 hours) for biochemical analysis, including serum albumin, total protein, hemoglobin, serum creatinine, blood urea nitrogen, serum calcium, phosphorus, parathyroid hormone, and other relevant parameters as clinically indicated.

Data Management and Statistical Analysis

All collected data were entered into a structured database using Microsoft Excel and subsequently analyzed using statistical software (IBM SPSS version 20.0). Data entry was performed by trained personnel with double-entry verification for critical variables to minimize transcription errors. Range and consistency checks were performed to identify and correct any data entry errors before analysis. Continuous variables were described using measures of central tendency (mean, median) and dispersion (standard deviation, interquartile range) after assessment of distribution normality using the Kolmogorov-Smirnov test. Categorical variables were described using frequencies and percentages. For comparative analysis, appropriate parametric (Student's t-test, ANOVA) or non-parametric tests (Mann-Whitney U test, Kruskal-Wallis test) were used based on data distribution characteristics. Correlation analysis was performed using Pearson's or Spearman's correlation coefficients as appropriate to examine relationships between nutritional parameters and growth outcomes. Multiple regression analysis was planned to identify independent predictors of growth outcomes while controlling for potential confounding variables. Statistical significance was set at $p < 0.05$ for all analyses, and results were presented with appropriate confidence intervals where applicable.

Ethical Considerations

The study protocol was submitted to and approved by the Institutional Ethics Committee of People's College of Medical Sciences & Research Centre, Bhopal, prior to commencement of data collection. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and Good Clinical Practice guidelines. Written informed consent was obtained from parents or legal guardians of all participating children after providing detailed information about the study objectives, procedures, potential benefits, and risks in the local language. For children above 7 years of age, age-appropriate assent was also obtained after explaining the study procedures in simple terms.

Results

Table 1: Demographic and Clinical Characteristics of Study Population (n=100)

Parameter		**Frequency (%) / Mean ± SD**
Age Distribution	2-5 years	24 (24.0)
	6-10 years	32 (32.0)
	11-15 years	28 (28.0)
	16-18 years	16 (16.0)
Mean age (years)		9.2 ± 4.1
Gender	Male	62 (62.0)
	Female	38 (38.0)
CKD Stage	Stage 2 (GFR 60-89)	18 (18.0)
	Stage 3 (GFR 30-59)	34 (34.0)
	Stage 4 (GFR 15-29)	28 (28.0)

	Stage 5 (GFR <15)	20 (20.0)
Primary Renal Diagnosis	CAKUT	52 (52.0)
	Glomerulonephritis	24 (24.0)
	Hereditary nephritis	12 (12.0)
	Polycystic kidney disease	8 (8.0)
	Others	4 (4.0)
Duration of CKD (months)		28.4 ± 18.6

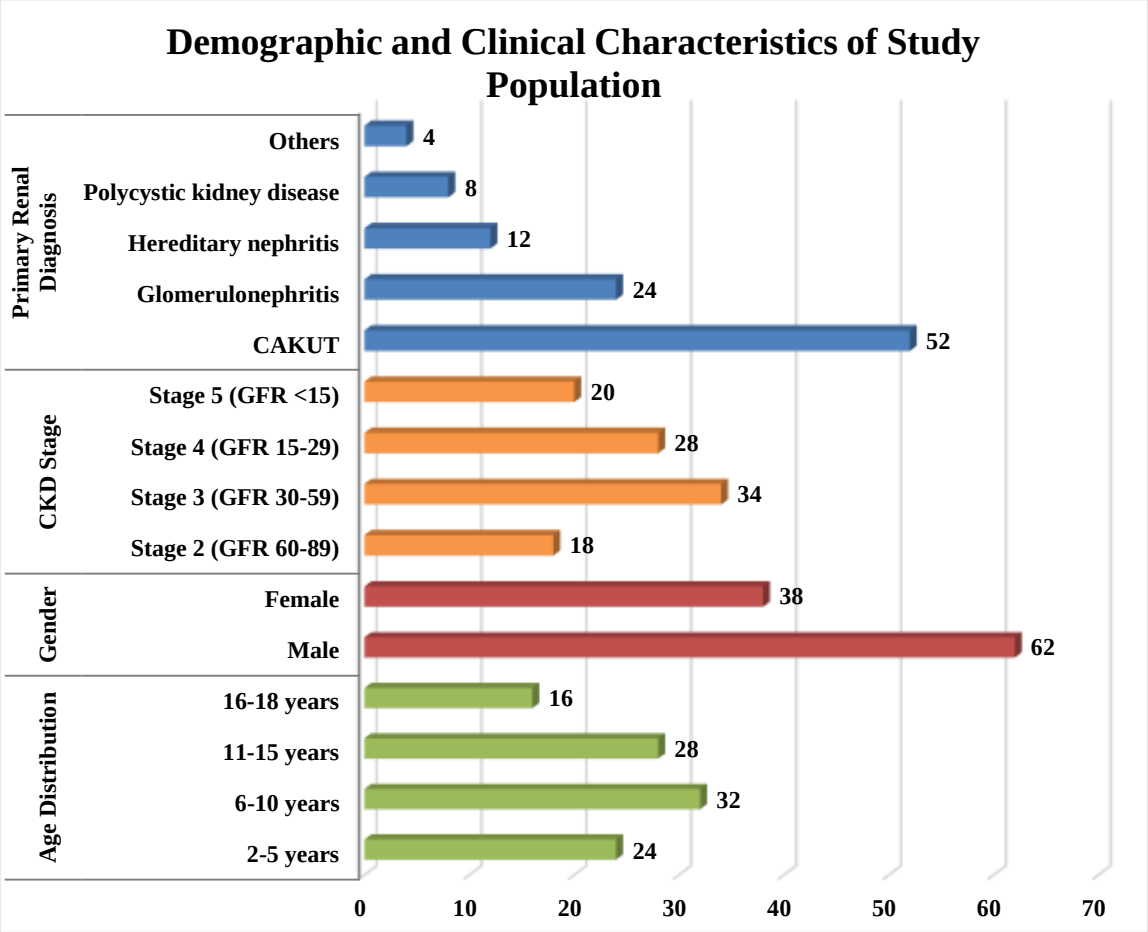

Fig: 1

Table 2: Anthropometric Measurements and Growth Parameters (n=100)

Parameter	Mean ± SD	Normal Range n(%)	Below Normal n(%)
Height-for-age Z-score	-2.1 ± 1.4	31 (31.0)	69 (69.0)
Weight-for-age Z-score	-1.8 ± 1.2	42 (42.0)	58 (58.0)
BMI-for-age Z-score	-1.3 ± 1.1	64 (64.0)	36 (36.0)
Growth Failure (<-2 SD)			
Stunting (Height-for-age)			45 (45.0)
Underweight (Weight-for-age)			38 (38.0)
Wasting (BMI-for-age)			22 (22.0)
Head Circumference Z-score (n=56, <5 years)	-1.6 ± 1.3	21 (37.5)	35 (62.5)
MUAC (cm)	17.2 ± 3.4	58 (58.0)	42 (42.0)
Triceps Skinfold (mm)	8.4 ± 3.2	51 (51.0)	49 (49.0)

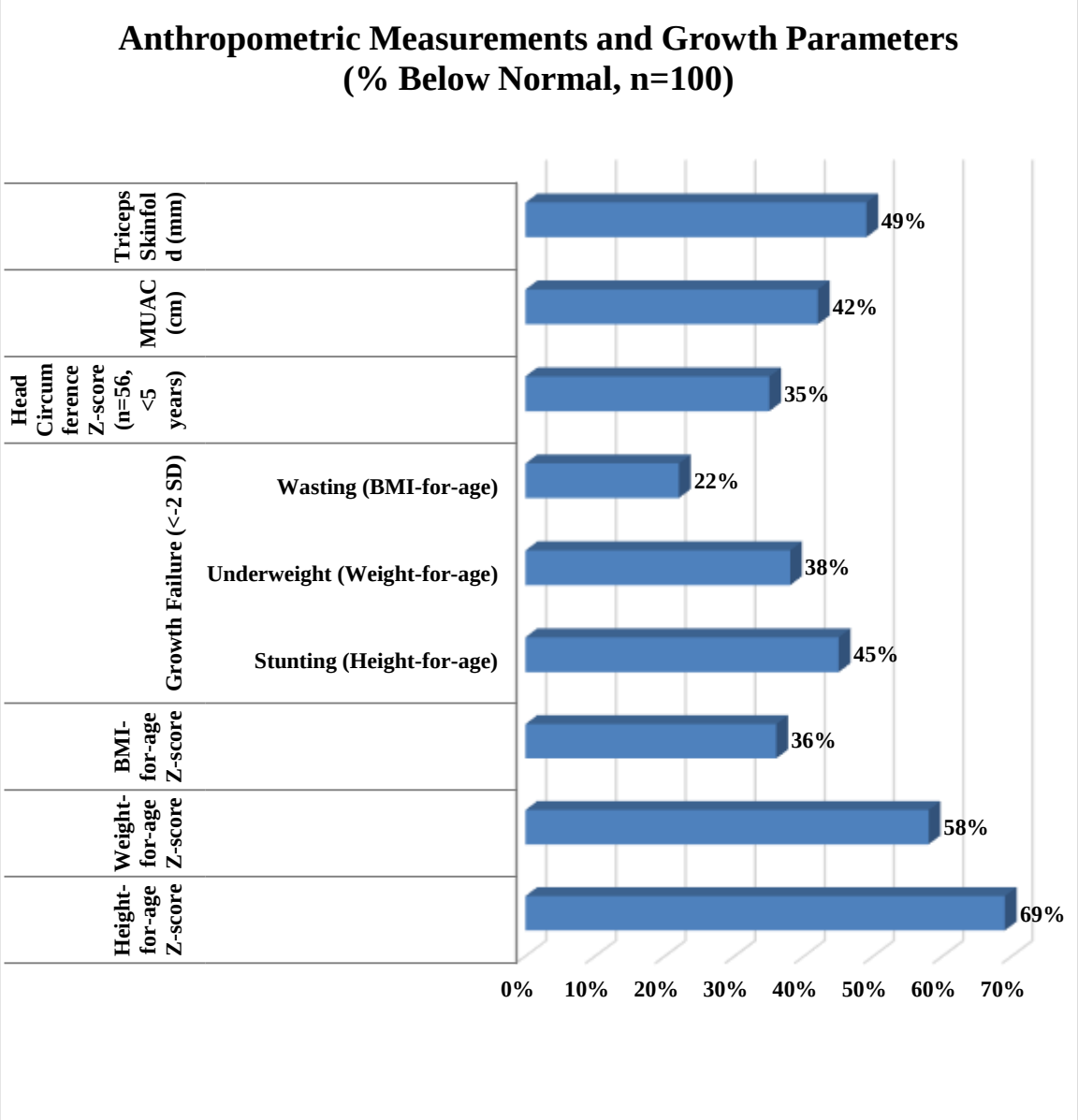

Fig: 2

Table 3: Nutritional Status Classification Based on Different Criteria (n=100)

Nutritional Status	Weight-for-Height n(%)	BMI-for-age n(%)	MUAC n(%)	Composite Assessment n(%)
Normal	56 (56.0)	64 (64.0)	58 (58.0)	34 (34.0)
Mild Malnutrition	28 (28.0)	22 (22.0)	26 (26.0)	41 (41.0)
Moderate Malnutrition	12 (12.0)	11 (11.0)	13 (13.0)	19 (19.0)
Severe Malnutrition	4 (4.0)	3 (3.0)	3 (3.0)	6 (6.0)
CKD Stage Distribution of Malnutrition				
Stage 2	6/18 (33.3)	4/18 (22.2)	5/18 (27.8)	8/18 (44.4)
Stage 3	13/34 (38.2)	11/34 (32.4)	12/34 (35.3)	21/34 (61.8)
Stage 4	16/28 (57.1)	14/28 (50.0)	15/28 (53.6)	22/28 (78.6)
Stage 5	9/20 (45.0)	7/20 (35.0)	10/20 (50.0)	15/20 (75.0)

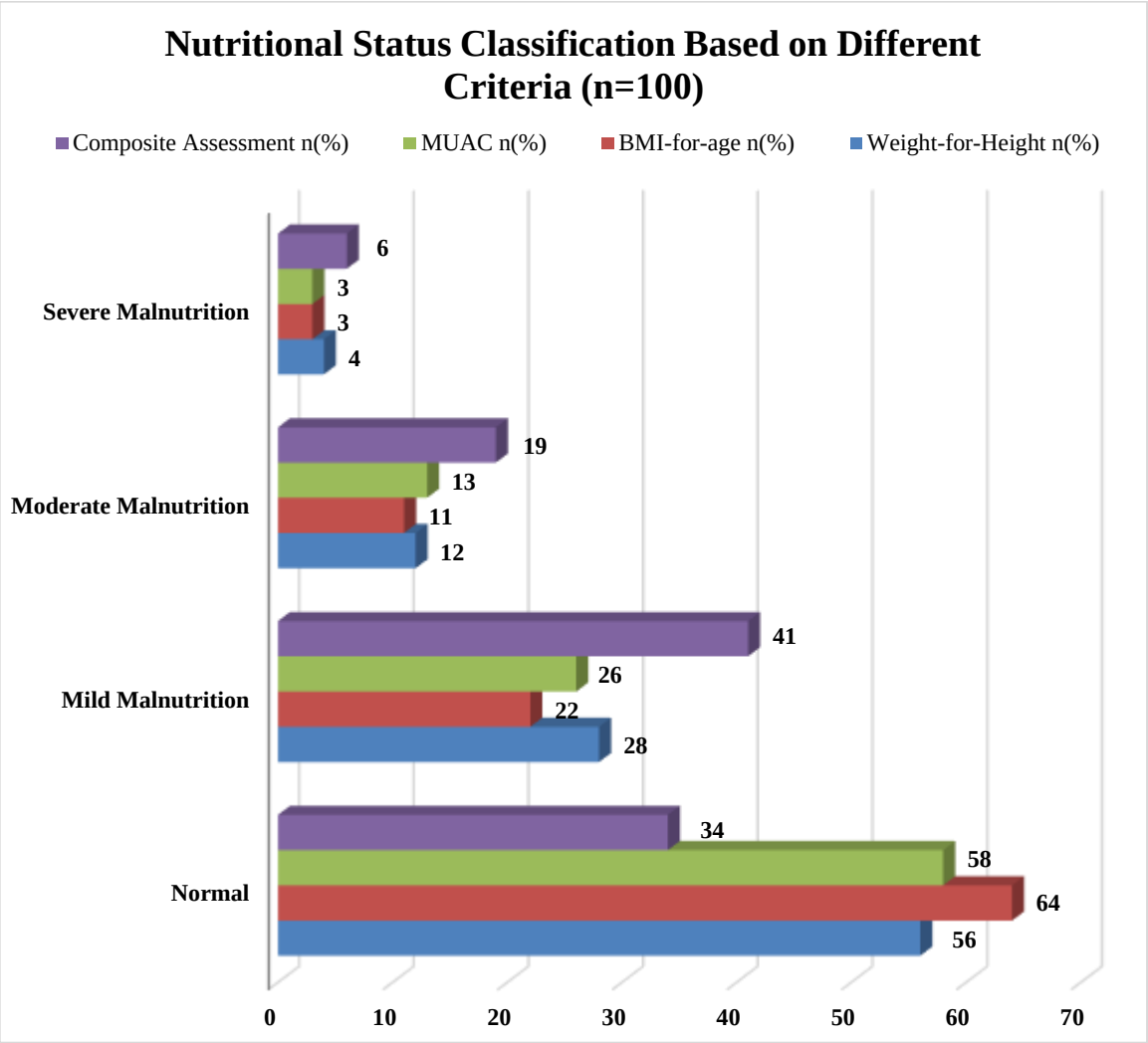

Fig: 3

Table 4: Biochemical Parameters According to CKD Stages (n=100)

Parameter	Stage 2 (n=18)	Stage 3 (n=34)	Stage 4 (n=28)	Stage 5 (n=20)	Normal Range
Serum Albumin (g/dL)	4.1 ± 0.4	3.8 ± 0.5	3.5 ± 0.6	3.2 ± 0.7	3.5-5.0
Hemoglobin (g/dL)	11.8 ± 1.2	10.6 ± 1.4	9.4 ± 1.3	8.2 ± 1.5	11.0-14.0
Serum Creatinine (mg/dL)	1.2 ± 0.3	2.1 ± 0.6	3.8 ± 1.2	6.4 ± 2.1	0.3-1.0
BUN (mg/dL)	28 ± 8	45 ± 12	72 ± 18	98 ± 24	8-20
Serum Calcium (mg/dL)	9.6 ± 0.4	9.2 ± 0.6	8.8 ± 0.7	8.4 ± 0.8	8.5-10.5
Serum Phosphorus (mg/dL)	4.2 ± 0.6	4.8 ± 0.8	5.4 ± 1.1	6.2 ± 1.4	3.0-4.5
PTH (pg/mL)	68 ± 22	124 ± 38	248 ± 78	412 ± 126	10-65
25(OH) Vitamin D (ng/mL)	18.4 ± 6.2	16.2 ± 5.8	14.6 ± 5.1	12.8 ± 4.6	30-100

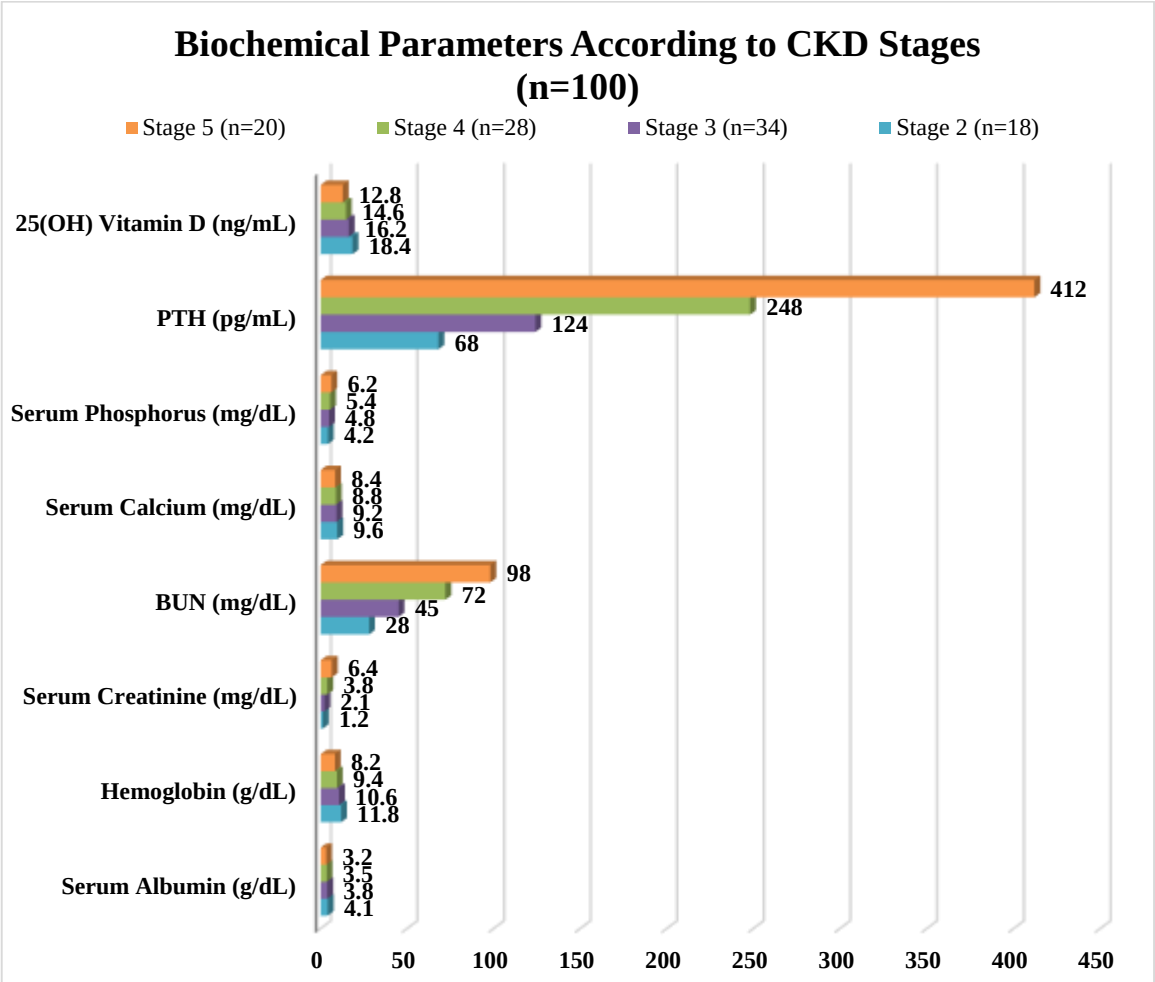

Fig: 4

Table 5: Dietary Assessment Results Compared to Recommended Intake (n=100)

Parameter	Mean Intake ± SD	Recommended Intake	% Meeting Requirements n(%)
Energy Intake			
Calories/kg/day	68.2 ± 24.6	80-100	34 (34.0)
Total calories/day	1486 ± 412	Age-appropriate	28 (28.0)
Protein Intake			
Protein g/kg/day	1.8 ± 0.6	2.0-2.5	42 (42.0)
Total protein g/day	46.8 ± 18.2	Age-appropriate	38 (38.0)
Micronutrient Intake			
Iron (mg/day)	8.4 ± 3.2	10-15	31 (31.0)
Calcium (mg/day)	486 ± 186	800-1200	18 (18.0)
Phosphorus (mg/day)	642 ± 224	500-1250	76 (76.0)
Dietary Adequacy by CKD Stage			
Stage 2: Adequate intake			11/18 (61.1)
Stage 3: Adequate intake			16/34 (47.1)
Stage 4: Adequate intake			8/28 (28.6)
Stage 5: Adequate intake			4/20 (20.0)

Table 6: Correlation Analysis Between Nutritional Parameters and Growth Outcomes (n=100)

Variables	Height Z-score	Weight Z-score	BMI Z-score	MUAC
Serum Albumin	0.64**	0.58**	0.42**	0.51**
Hemoglobin	0.56**	0.52**	0.38**	0.46**
Energy Intake (cal/kg)	0.48**	0.61**	0.55**	0.49**
Protein Intake (g/kg)	0.52**	0.58**	0.47**	0.54**
CKD Stage	-0.41**	-0.38**	-0.28*	-0.35**
Duration of CKD	-0.33**	-0.29*	-0.21*	-0.26*
PTH Level	-0.39**	-0.34**	-0.24*	-0.31**
Serum Phosphorus	-0.35**	-0.32**	-0.22*	-0.28*
25(OH) Vitamin D	0.31**	0.28*	0.19	0.24*
Age at CKD onset	-0.45**	-0.39**	-0.26*	-0.33**

*$p < 0.05$; **$p < 0.01$

Discussion

The present study revealed a high prevalence of growth failure among children with chronic kidney disease, with 69% of patients demonstrating height-for-age Z-scores below normal ranges and 45% showing frank stunting (height-for-age Z-score <-2 SD). These findings are consistent with previous reports from both developed and developing countries, though the prevalence observed in our study appears to be on the higher end of the reported range. Seikaly et al. (2006) analyzed data from the North American Pediatric Renal Transplant Cooperative Study (NAPRTCS) and found that approximately 37% of children with CKD had heights below the 5th percentile, while our study found a higher prevalence of 45% with severe stunting. This difference may reflect the later presentation and more advanced disease stage at diagnosis commonly observed in developing countries, as well as potential differences in nutritional management and socioeconomic factors affecting our study population.

The strong correlation between CKD stage and growth parameters observed in our study ($r = -0.41$ for height Z-score, $p < 0.01$) aligns with the established understanding that growth failure becomes more pronounced with declining renal function. Norman et al. (2000) similarly demonstrated that growth retardation was significantly related to the severity of renal impairment, with children having more severe CKD showing greater growth deficits. The mean height-for-age Z-score of -2.1 ± 1.4 in our study population indicates that the average child was significantly shorter than age-matched healthy peers, emphasizing the substantial impact of CKD on linear growth in the pediatric population.

Interestingly, our data showed a relatively lower prevalence of wasting (22%) compared to stunting (45%), suggesting that linear growth is more severely affected than weight gain in children with CKD. This pattern is consistent with the chronic nature of growth failure in CKD, where long-term nutritional and metabolic disturbances primarily affect height velocity rather than acute weight loss. The preservation of weight relative to height may also indicate compensatory mechanisms or clinical interventions that help maintain caloric intake despite the underlying disease process.

The composite nutritional assessment in our study revealed that 66% of children with CKD had some degree of malnutrition, with 6% showing severe malnutrition. This prevalence is higher than that reported in many developed countries but consistent with studies from other developing nations. The progressive increase in malnutrition prevalence with advancing CKD stages (44.4% in Stage 2 to 75.0% in Stage 5) demonstrates the close relationship between declining renal function and nutritional deterioration.

Serum albumin levels showed a significant inverse correlation with CKD stage and strong positive correlations with all growth parameters ($r = 0.64$ for height Z-score, $p < 0.01$). The mean albumin levels ranged from 4.1 ± 0.4 g/dL in Stage 2 to 3.2 ± 0.7 g/dL in Stage 5, indicating progressive protein-energy wasting with disease advancement. Foster and Leonard (2004) emphasized that

while serum albumin is influenced by factors other than nutritional status in CKD patients, it remains a valuable marker when interpreted in the clinical context. The strong correlations observed in our study suggest that albumin continues to be a useful indicator of nutritional status in pediatric CKD patients.

Anemia was highly prevalent across all CKD stages, with mean hemoglobin levels declining from 11.8 ± 1.2 g/dL in Stage 2 to 8.2 ± 1.5 g/dL in Stage 5. The significant correlation between hemoglobin levels and growth parameters ($r = 0.56$ for height Z-score, $p < 0.01$) suggests that anemia may contribute to growth failure through multiple mechanisms, including reduced oxygen delivery to tissues, decreased appetite, and impaired physical activity. Previous studies have shown that correction of anemia in children with CKD can lead to improvements in growth velocity, supporting the importance of addressing this complication as part of comprehensive nutritional management.

The progressive deterioration in mineral metabolism observed in our study population reflects the well-established pathophysiology of CKD-mineral and bone disorder (CKD-MBD). Parathyroid hormone (PTH) levels showed a dramatic increase across CKD stages, from 68 ± 22 pg/mL in Stage 2 to 412 ± 126 pg/mL in Stage 5, far exceeding normal ranges. The significant negative correlation between PTH levels and growth parameters ($r = -0.39$ for height Z-score, $p < 0.01$) supports the hypothesis that secondary hyperparathyroidism contributes to growth failure through direct effects on the growth plate and interference with growth hormone action.

Vitamin D deficiency was universal across all CKD stages in our study population, with mean 25(OH) vitamin D levels ranging from 18.4 ng/mL in Stage 2 to 12.8 ng/mL in Stage 5, all well below the recommended minimum of 30 ng/mL. This finding is consistent with reports from other Indian studies showing high prevalence of vitamin D deficiency in both healthy children and those with CKD. The positive correlation between vitamin D levels and growth parameters, though modest ($r = 0.31$ for height Z-score, $p < 0.01$), suggests that vitamin D deficiency may contribute to growth failure through both direct effects on bone metabolism and indirect effects through secondary hyperparathyroidism.

Hyperphosphatemia became increasingly prevalent with advancing CKD stages, with mean phosphorus levels exceeding normal ranges in Stages 4 and 5. The negative correlation between serum phosphorus and growth parameters may reflect both the severity of underlying CKD and the potential growth-inhibiting effects of phosphorus retention. Mehls et al. (2008) emphasized that optimal management of mineral metabolism disorders is crucial for maximizing growth potential in children with CKD.

The dietary assessment revealed significant inadequacies in energy and protein intake across the study population, with only 34% of children meeting recommended caloric requirements and 42% meeting protein recommendations. The mean energy intake of 68.2 ± 24.6 calories/kg/day was substantially below the recommended 80-100 calories/kg/day for children with CKD, indicating widespread dietary inadequacy that could contribute to poor growth outcomes. The strong positive correlations between energy intake and growth parameters ($r = 0.48$ for height Z-score, $r = 0.61$ for weight Z-score, both $p < 0.01$) underscore the critical importance of adequate caloric intake for normal growth in children with CKD.

Protein intake showed similar patterns of inadequacy, with a mean intake of 1.8 ± 0.6 g/kg/day falling below the recommended 2.0-2.5 g/kg/day for children with CKD. The KDOQI guidelines (2009) emphasize that children with CKD require higher protein intake than healthy children to compensate for uremia-induced protein catabolism and to support normal growth and development. The significant correlation between protein intake and growth parameters ($r = 0.52$ for height Z-score, $p < 0.01$) confirms the importance of adequate protein provision in this population.

Micronutrient deficiencies were common, particularly for calcium (only 18% meeting requirements) and iron (31% meeting requirements). The low calcium intake, combined with high phosphorus intake in some patients, may contribute to the development of secondary hyperparathyroidism and

bone disease. These findings highlight the need for specialized nutritional counseling and potentially nutritional supplementation in children with CKD.

The negative correlation between duration of CKD and growth parameters ($r = -0.33$ for height Z-score, $p < 0.01$) suggests that prolonged exposure to the uremic environment results in cumulative growth deficits. More importantly, the strong negative correlation between age at CKD onset and growth outcomes ($r = -0.45$ for height Z-score, $p < 0.01$) confirms that children who develop CKD at younger ages experience more severe growth impairment. This finding is consistent with previous studies showing that early-onset CKD has more devastating effects on final adult height, as it affects growth during the most critical periods of childhood development.

Kari et al. (2000) demonstrated that infants with severe chronic renal failure had particularly poor growth outcomes, with many failing to achieve normal height despite optimal medical management. Our findings extend this observation to the broader pediatric CKD population, showing that early disease onset continues to be a significant risk factor for growth failure across all age groups.The comprehensive assessment of nutritional status and growth patterns in our study population reveals multiple interconnected factors contributing to poor outcomes in children with CKD. The strong correlations between biochemical markers, dietary intake, and growth parameters suggest that interventions targeting multiple aspects of nutritional management may be more effective than addressing individual deficiencies in isolation.

The high prevalence of growth failure and malnutrition observed in our study, particularly in advanced CKD stages, emphasizes the need for early and aggressive nutritional intervention. The KDOQI guidelines (2009) recommend regular nutritional assessment and intervention beginning in CKD Stage 2, but our findings suggest that even earlier intervention may be beneficial, particularly for children diagnosed at young ages.

The universal vitamin D deficiency and high prevalence of secondary hyperparathyroidism in our study population highlight the importance of addressing mineral metabolism disorders as part of comprehensive growth management. The significant correlations between PTH levels and growth parameters suggest that optimal management of CKD-MBD may have direct benefits on growth outcomes beyond the prevention of bone disease.

Conclusion

This cross-sectional study of 100 children with chronic kidney disease revealed a high prevalence of growth failure (69%) and malnutrition (66%), with both conditions becoming more severe with advancing CKD stages. Stunting (45%) was more common than wasting (22%), indicating that linear growth is predominantly affected in pediatric CKD. Strong positive correlations were observed between serum albumin, hemoglobin, energy intake, protein intake, and growth parameters, while CKD stage, PTH levels, and early disease onset showed significant negative correlations with growth outcomes. Biochemical abnormalities including anemia, secondary hyperparathyroidism, and vitamin D deficiency were universal across all CKD stages. Dietary assessment revealed inadequate energy (66% below requirements) and protein intake (58% below requirements) in the majority of patients. The findings demonstrate that growth failure in pediatric CKD results from complex interactions between declining renal function, nutritional inadequacy, mineral metabolism disorders, and chronic inflammation, with children developing CKD at younger ages being particularly vulnerable to severe growth impairment.

Recommendations

Early and comprehensive nutritional intervention should be implemented beginning in CKD Stage 2, with regular monitoring of growth parameters, nutritional status, and biochemical markers every 3-6 months. Specialized dietary counseling should focus on achieving adequate energy (80-100 cal/kg/day) and protein intake (2.0-2.5 g/kg/day) through culturally appropriate food choices and nutritional supplementation when necessary. Aggressive management of anemia, secondary hyperparathyroidism, and vitamin D deficiency should be prioritized as integral components of

growth optimization strategies. Growth hormone therapy should be considered for children with persistent growth failure despite optimal nutritional and medical management, particularly those with early-onset CKD. Healthcare providers should receive training in pediatric CKD nutrition management, and standardized protocols should be developed for nutritional assessment and intervention. Future research should focus on developing cost-effective nutritional interventions suitable for resource-limited settings and evaluating the long-term impact of early nutritional optimization on adult height and quality of life. Family education programs should be established to improve dietary compliance and recognition of nutritional problems, while policy initiatives should address food security and access to specialized nutritional products for children with CKD.

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