

Nutritional Status and its Association with Disease Progression in Chronic Kidney Disease: Prospective Observational Study

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Abstract:

Background: Chronic kidney disease (CKD) is a progressive disorder associated with substantial morbidity and mortality. Nutritional abnormalities are common during CKD and may include reduced dietary intake, loss of muscle mass, low body weight, hypoalbuminemia and protein-energy wasting. Nutritional assessment is an important component of comprehensive CKD care.

Objectives: To assess nutritional status among adults with CKD and to evaluate its association with disease progression over a 12-month follow-up period.

Methods: This prospective observational cohort study included 500 adults with established CKD attending Patna Medical College and Hospital, Patna, Bihar. Nutritional status was assessed using body mass index (BMI), serum albumin, dietary protein intake and the Malnutrition-Inflammation Score (MIS). Participants were followed for 12 months. CKD progression was defined as a sustained $\geq 40\%$ decline in eGFR from baseline or progression to kidney failure. Associations were assessed using categorical tests and multivariable logistic regression.

Results: The mean age was 52.6 ± 12.7 years and 280 (56.0%) participants were male. Underweight status was present in 110 (22.0%), serum albumin < 3.5 g/dL in 145 (29.0%), dietary protein intake < 0.6 g/kg/day in 150 (30.0%), and MIS ≥ 8 in 95 (19.0%). During 12 months, 120 (24.0%) participants met the predefined criteria for CKD progression. Progression was more frequent among participants with low BMI, low serum albumin, lower protein intake and higher MIS. In multivariable analysis, MIS ≥ 8 , albumin < 3.5 g/dL, BMI < 18.5 kg/m² and protein intake < 0.6 g/kg/day were independently associated with progression.

Conclusion: Impaired nutritional status was associated with a higher frequency of CKD progression in this cohort. Early identification of nutritional abnormalities and individualized nutritional management may have an important role in comprehensive CKD care.

Keywords: Chronic Kidney Disease; Nutritional Status; Protein-Energy Wasting; Malnutrition-Inflammation Score; BMI; Serum Albumin; eGFR; Disease Progression.

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Introduction

Chronic kidney disease (CKD) is a major global health problem characterized by persistent abnormalities of kidney structure or function with implications for health. The clinical course is heterogeneous, and identification of individuals at increased risk of progression is central to CKD management. [1]

Nutritional abnormalities are an important component of CKD. Reduced appetite, dietary restrictions, inflammation, metabolic acidosis, hormonal disturbances and altered protein metabolism may contribute to loss of body protein and energy stores. Current nutrition guidance emphasizes systematic nutritional assessment and

individualized medical nutrition therapy in CKD. [2] Protein-energy wasting (PEW) is a multifactorial syndrome associated with CKD and adverse clinical outcomes. [3,4]

Nutritional assessment in CKD is challenging because no single marker completely reflects nutritional status. BMI, weight change, dietary intake, biochemical parameters, anthropometric measurements and composite nutritional scores provide complementary information. The Malnutrition-Inflammation Score (MIS) has been evaluated in nondialysis CKD and is associated with nutritional and inflammatory abnormalities and clinical outcomes. [5,6]

The relationship between nutrition and CKD progression may be bidirectional. Inadequate energy and protein intake can contribute to muscle and fat loss, while worsening CKD may itself cause anorexia, inflammation and metabolic disturbances. [3,4]

Current KDIGO guidance recommends maintaining an appropriate protein intake in adults with CKD while avoiding excessive protein intake, with attention to the risk of undernutrition and wasting. [1] The KDOQI guideline similarly emphasizes systematic nutritional assessment and individualized medical nutrition therapy. [2]

Therefore, this study evaluated nutritional status among adults with CKD and examined its association with CKD progression over 12 months.

Objectives

1. To assess nutritional status among adults with chronic kidney disease attending PMCH, Patna.
2. To determine the frequency of nutritional abnormalities based on BMI, serum albumin, dietary protein intake and MIS.
3. To assess the relationship between nutritional status and CKD stage.
4. To determine the proportion of participants demonstrating CKD progression during 12 months of follow-up.
5. To evaluate the association between baseline nutritional abnormalities and subsequent CKD progression.
6. To identify nutritional factors independently associated with CKD progression.

Materials and Methods

Study Design: This was a prospective observational cohort study conducted among adults with established CKD.

Study Setting: The study was conducted in the Department of Nephrology/Medicine at Patna Medical College and Hospital (PMCH), Patna, Bihar, India.

Study Duration: The study duration was 12 months.

Study Population: Adults aged ≥ 18 years with established CKD attending the study centre during the recruitment period were eligible for assessment.

Sample Size: The required sample size was estimated using the single-population proportion formula:

$$n = Z^2 \times p \times (1 - p) / d^2$$

where Z is the standard normal deviate corresponding to a 95% confidence level (1.96), p is the anticipated proportion (0.20), and d is the absolute precision (0.04).

$$n = (1.96^2 \times 0.20 \times 0.80) / (0.04^2) = 384.16$$

The calculated sample size was rounded to 385 participants. A 30% inflation factor was then applied:

$385 \times 1.30 = 500.5$, rounded to 500 participants. The final analysed cohort comprised 500 participants.

Inclusion Criteria: Age ≥ 18 years; established CKD; attendance at the study centre during the study period; willingness to participate and complete nutritional assessment and follow-up; and adequate baseline clinical and laboratory information.

Exclusion Criteria: Acute kidney injury without established CKD; active malignancy likely to substantially alter nutritional status; acute severe infection at baseline; pregnancy; severe liver disease; inability to complete nutritional assessment; or incomplete baseline information required for the primary analysis.

Assessment of Nutritional Status: Nutritional status was assessed using multiple complementary parameters rather than a single measurement, consistent with CKD nutrition guidance. [2]

BMI was calculated as weight (kg) divided by height squared (m^2). BMI categories were <18.5 , $18.5-24.9$, $25.0-29.9$ and ≥ 30 kg/m^2 .

Serum albumin was measured using the institutional laboratory method and categorized as <3.5 g/dL or ≥ 3.5 g/dL for analysis.

Dietary protein intake was estimated using dietary assessment and expressed as g/kg/day. Categories used for analysis were <0.6 , $0.6-0.8$ and >0.8 g/kg/day.

MIS was used as a composite assessment of nutritional and inflammatory status. For this analysis, MIS ≥ 8 was categorized as increased nutritional risk.

Assessment of Kidney Function: Serum creatinine was measured using the institutional laboratory method. eGFR was estimated using the CKD-EPI approach consistent with the laboratory and study-period reporting standards. CKD stage was classified according to eGFR categories. [1]

Definition of CKD Progression: For this study, CKD progression was defined as a sustained $\geq 40\%$ decline in eGFR from baseline or progression to kidney failure, including sustained eGFR <15 mL/min/1.73 m^2 or initiation of kidney replacement therapy.

Follow-up: Participants were followed for 12 months. Clinical assessment, serum creatinine and relevant laboratory investigations were repeated at scheduled follow-up visits. Baseline and final kidney function were compared to determine progression.

Statistical Analysis: Continuous variables were summarized as mean $\pm$ standard deviation or median with interquartile range as appropriate. Categorical variables were summarized as frequencies and percentages. Associations between categorical nutritional variables and CKD progression were assessed using the chi-square test or Fisher's exact test where appropriate. Continuous variables were compared using the independent-samples t-test or Mann–Whitney U test according to distribution. Multivariable logistic regression was used to identify factors independently associated with CKD progression. Adjusted odds ratios with 95% confidence intervals were reported. A two-sided p

value <0.05 was considered statistically significant.

Reporting Guidelines: The study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for observational studies [10].

Results

A total of 500 participants with established CKD were included. The mean age was 52.6 ± 12.7 years, with a range of 19–79 years, and 280 (56.0%) participants were male. The distribution of demographic and clinical characteristics is shown in Table 1.

Table 1: Baseline demographic and clinical characteristics of the study population

Characteristic	Number (n=500)	Percentage
Age 18–39 years	82	16.4%
Age 40–59 years	238	47.6%
Age ≥ 60 years	180	36.0%
Male	280	56.0%
Female	220	44.0%
CKD G2	55	11.0%
CKD G3a	120	24.0%
CKD G3b	145	29.0%
CKD G4	115	23.0%
CKD G5	65	13.0%
Diabetes mellitus	248	49.6%
Hypertension	372	74.4%
Cardiovascular disease	118	23.6%

Nutritional assessment demonstrated that 110 (22.0%) participants were underweight, 210 (42.0%) had normal BMI, 120 (24.0%) were overweight and 60 (12.0%) were obese. Serum albumin was <3.5 g/dL in 145 (29.0%) participants. Dietary protein intake was <0.6 g/kg/day in 150 (30.0%)

participants, while 215 (43.0%) had intake of 0.6–0.8 g/kg/day and 135 (27.0%) had intake >0.8 g/kg/day. MIS ≥ 8 was observed in 95 (19.0%) participants. The distribution is presented in Table 2 and illustrated in Figure 1.

Table 2: Distribution of nutritional parameters among study participants

Nutritional parameter	Number	Percentage
BMI <18.5 kg/m ²	110	22.0%
BMI 18.5–24.9 kg/m ²	210	42.0%
BMI 25.0–29.9 kg/m ²	120	24.0%
BMI ≥ 30 kg/m ²	60	12.0%
Albumin <3.5 g/dL	145	29.0%
Albumin ≥ 3.5 g/dL	355	71.0%
Protein <0.6 g/kg/day	150	30.0%
Protein 0.6–0.8 g/kg/day	215	43.0%
Protein >0.8 g/kg/day	135	27.0%
MIS <8	405	81.0%
MIS ≥ 8	95	19.0%

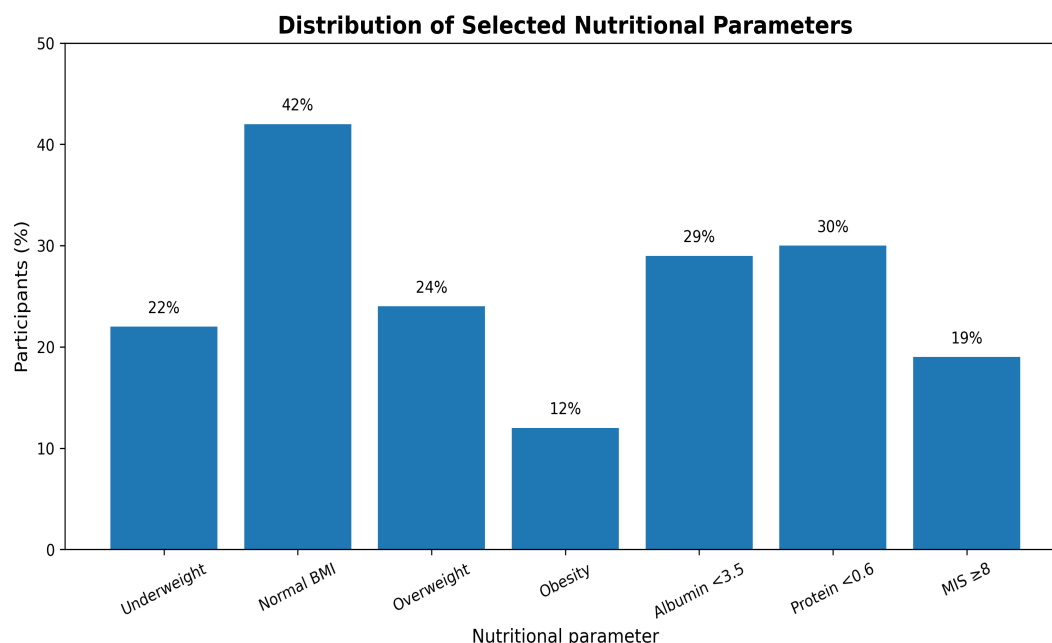


Figure 1: Distribution of selected nutritional parameters among the study population.

During 12 months of follow-up, 120 (24.0%) participants fulfilled the predefined criteria for CKD progression, while 380 (76.0%) did not. Of those with progression, 113 met the sustained $\geq 40\%$ eGFR decline criterion and 7 progressed to kidney failure.

The mean baseline eGFR was 38.6 ± 17.2 mL/min/1.73 m² and the 12-month eGFR was 34.7 ± 16.8 mL/min/1.73 m². Progression by baseline CKD stage is shown in Table 3 and Figure 2.

Table 3: CKD progression according to baseline CKD stage

CKD stage	Total	Progression	No progression	Progression (%)
G2	55	8	47	14.5%
G3a	120	17	103	14.2%
G3b	145	31	114	21.4%
G4	115	36	79	31.3%
G5	65	28	37	43.1%
Total	500	120	380	24.0%

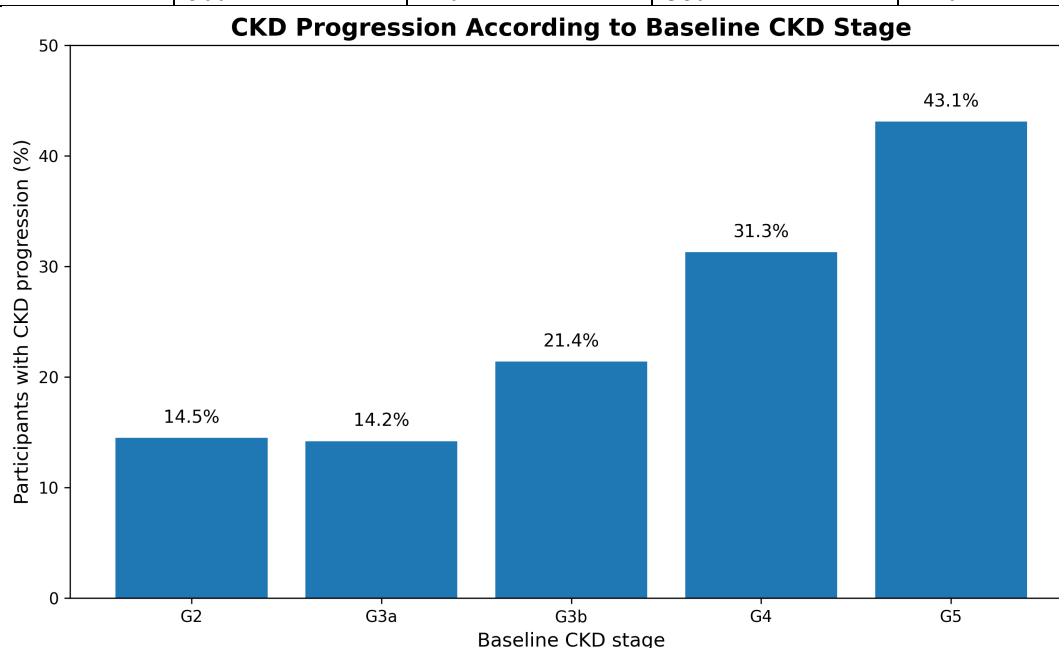


Figure 2: Proportion of participants demonstrating CKD progression according to baseline CKD stage.

CKD progression occurred in 45 (40.9%) underweight participants, 42 (20.0%) participants with normal BMI, 23 (19.2%) overweight participants and 10 (16.7%) participants with obesity. The association between BMI category and progression was statistically significant ($\chi^2=22.39$, $p<0.001$). Progression occurred in 56 (38.6%) participants with albumin <3.5 g/dL compared with 64 (18.0%) with albumin ≥ 3.5 g/dL ($\chi^2=22.82$,

$p<0.001$). Progression was observed in 55 (36.7%) participants with protein intake <0.6 g/kg/day, 45 (20.9%) with intake $0.6-0.8$ g/kg/day and 20 (14.8%) with intake >0.8 g/kg/day ($\chi^2=20.55$, $p<0.001$). Among participants with MIS ≥ 8 , 45 (47.4%) experienced progression compared with 75 (18.5%) with MIS <8 ($\chi^2=33.55$, $p<0.001$). These findings are summarized in Table 4.

Table 4: Association between nutritional parameters and CKD progression

Nutritional parameter	Progression n (%)	No progression n (%)	p value
BMI <18.5 kg/m ²	45 (40.9%)	65 (59.1%)	<0.001
BMI $18.5-24.9$ kg/m ²	42 (20.0%)	168 (80.0%)	
BMI $25.0-29.9$ kg/m ²	23 (19.2%)	97 (80.8%)	
BMI ≥ 30 kg/m ²	10 (16.7%)	50 (83.3%)	
Albumin <3.5 g/dL	56 (38.6%)	89 (61.4%)	<0.001
Albumin ≥ 3.5 g/dL	64 (18.0%)	291 (82.0%)	
Protein <0.6 g/kg/day	55 (36.7%)	95 (63.3%)	<0.001
Protein $0.6-0.8$ g/kg/day	45 (20.9%)	170 (79.1%)	
Protein >0.8 g/kg/day	20 (14.8%)	115 (85.2%)	
MIS ≥ 8	45 (47.4%)	50 (52.6%)	<0.001
MIS <8	75 (18.5%)	330 (81.5%)	

Multivariable Analysis: In multivariable logistic regression, MIS ≥ 8 , albumin <3.5 g/dL, BMI <18.5 kg/m² and protein intake <0.6 g/kg/day remained

independently associated with CKD progression after adjustment for selected clinical variables (Table 5).

Table 5: Multivariable logistic regression analysis of factors associated with CKD progression

Variable	Adjusted OR	95% CI	p value
Age ≥ 60 years	1.42	0.94–2.16	0.094
Male sex	1.18	0.79–1.77	0.417
Diabetes mellitus	1.58	1.04–2.40	0.032
Baseline eGFR <30	2.31	1.51–3.54	<0.001
BMI <18.5 kg/m ²	1.78	1.10–2.88	0.019
Albumin <3.5 g/dL	2.12	1.38–3.26	<0.001
Protein intake <0.6 g/kg/day	1.69	1.09–2.62	0.019
MIS ≥ 8	2.85	1.75–4.63	<0.001

Participants with higher nutritional risk demonstrated a greater decline in eGFR over 12 months. Mean eGFR declined by 2.5 mL/min/ 1.73 m² among participants with MIS <8 compared with

7.8 mL/min/ 1.73 m² among those with MIS ≥ 8 . Corresponding changes according to albumin category are shown in Table 6 and Figure 3.

Table 6: Mean change in eGFR according to nutritional risk

Nutritional category	Baseline eGFR, mean $\pm$ SD	12-month eGFR, mean $\pm$ SD	Mean decline
MIS <8	39.4 ± 17.1	36.9 ± 16.9	2.5
MIS ≥ 8	35.1 ± 16.5	27.3 ± 15.8	7.8
Albumin ≥ 3.5 g/dL	39.8 ± 17.0	37.0 ± 16.6	2.8
Albumin <3.5 g/dL	35.7 ± 16.8	29.6 ± 16.2	6.1

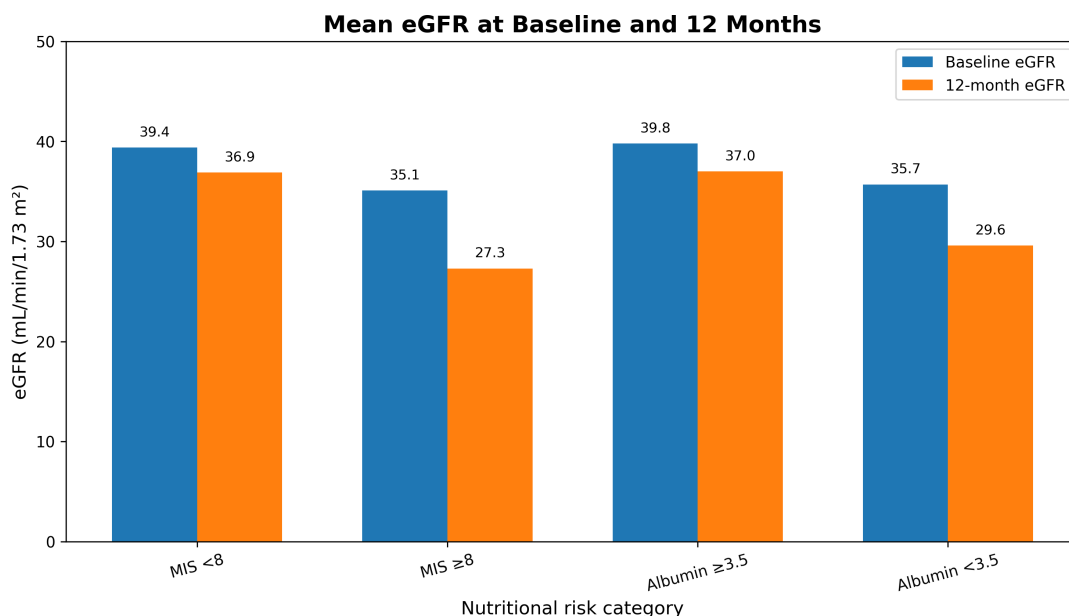


Figure 3: Mean eGFR at baseline and 12 months according to nutritional risk category.

Discussion

The present study evaluated nutritional status and its association with CKD progression over 12 months. The principal finding was that low BMI, hypoalbuminemia, lower dietary protein intake and higher MIS were associated with a higher frequency of CKD progression.

CKD is associated with metabolic and nutritional disturbances. Protein-energy wasting is multifactorial and reflects the interaction of inadequate intake, inflammation, metabolic acidosis, endocrine disturbances, comorbidity and catabolic processes. [3,4]

The observed nutritional abnormalities are consistent with the established burden of PEW in CKD. Indian hospital-based research has documented substantial nutritional impairment among adults with CKD. [7] A meta-analysis of contemporary observational studies likewise found that protein-energy wasting was common across kidney disease populations, including nondialysis CKD stages 3–5. [8]

Underweight status was associated with a higher frequency of progression in the present study. BMI is useful for screening but does not distinguish fat from muscle and can be influenced by fluid status; therefore, it should be interpreted together with other nutritional measures. Studies evaluating MIS have demonstrated associations with BMI, eGFR and biochemical markers of nutrition and inflammation. [5,9]

Low serum albumin was also associated with progression. Albumin is clinically useful but is influenced by inflammation, hydration, hepatic synthesis and other non-nutritional factors. It should

therefore be interpreted as one component of a broader nutritional assessment rather than as a standalone diagnostic marker. [2]

Lower dietary protein intake was associated with progression in this cohort. This finding should not be interpreted as evidence that higher protein intake is universally protective. KDIGO recommends approximately 0.8 g/kg/day in adults with CKD G3–G5 and advises avoiding high protein intake above 1.3 g/kg/day in adults at risk of progression, while emphasizing caution in people with sarcopenia, cachexia or undernutrition. [1]

The strong association between MIS and progression supports the potential value of composite nutritional assessment. Amparo et al. demonstrated that worsening MIS was associated with deterioration in nutritional and inflammatory indicators and with higher mortality risk in nondialysis CKD. [5] Jagadeswaran et al. similarly reported associations between higher MIS and adverse outcomes in predialysis CKD. [6]

Advancing CKD stage was associated with increasing progression in this cohort. This is clinically plausible because baseline kidney function is a major determinant of subsequent risk. Current KDIGO guidance emphasizes assessment of kidney function and albuminuria and individualized management of risk factors. [1]

The multivariable analysis suggested that MIS ≥8, low albumin, low BMI and low protein intake remained independently associated with progression. However, these findings do not prove causality. Worsening CKD can itself lead to anorexia, inflammation and nutritional decline, creating the possibility of reverse causality.

The clinical implication is that nutritional assessment should be incorporated into comprehensive CKD management. KDOQI recommends systematic nutritional assessment and individualized medical nutrition therapy, including appropriate dietary protein and energy management. [2] Nutritional intervention should avoid both excessive restriction and inadequate intake.

Overall, the findings support early identification of nutritional vulnerability in CKD. Longer prospective studies with standardized dietary assessment, body-composition measurements and repeated nutritional evaluations are needed to clarify the causal relationship between nutritional status and kidney disease progression.

Strengths:

Prospective follow-up allowed nutritional status to be assessed before the outcome.

Multiple nutritional indicators were evaluated.

Kidney function was assessed longitudinally.

A clinically meaningful CKD progression outcome was used.

Potential confounding variables were considered in multivariable analysis.

Limitations

The observational design does not establish causality.

BMI and serum albumin are imperfect standalone indicators of nutritional status.

Dietary protein intake may be affected by recall and measurement error.

The 12-month follow-up may be insufficient to capture all long-term kidney outcomes.

The hospital-based setting may limit generalizability.

Residual confounding may remain despite multivariable adjustment.

Conclusion

Impaired nutritional status was associated with a higher frequency of CKD progression over 12 months. Low BMI, low serum albumin, inadequate dietary protein intake and elevated MIS were associated with greater progression, with higher MIS showing the strongest nutritional association in the multivariable model. Nutritional assessment and individualized dietary management should form part of comprehensive CKD care. Because this is an observational study, the findings should be interpreted as associations rather than proof of causality.

Declarations

Ethics Approval: The study was reviewed and approved by the Institutional Ethics Committee of Patna Medical College and Hospital (PMCH), Patna, Bihar, India, before initiation of participant enrolment and data collection. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and applicable institutional requirements.

Funding

Competing Interests: The authors declare that they have no competing interests.

Data Availability: The underlying study data will be available from the corresponding author or institution subject to institutional policy, participant confidentiality and applicable data-protection requirements.

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