Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 08 May 2026
Revised 07 June 2026
Accepted 20 June 2026

Natural Resources for Human Health 2026, Vol. 6 (5): 844–852

KEYWORDS:
Diabetic Nephropathies; Glycated Hemoglobin; Nutrition Assessment; Nutritional Status; Serum Albumin; Malnutrition.

Immunonutritional Vulnerability Across HbA1c Categories in Diabetic Kidney Disease: A Cross-Sectional Study

Dian Vera Widiawaty[1,2*], Suryani As'ad[1,2], Andi Yasmin Syauki[1,2], Nurpudji Astuti Daud[1,2], Aminuddin[1], Haerani Rasyid[1,3]

[1]Department of Clinical Nutrition, Faculty of Medicine, Hasanuddin University, Makassar, South Sulawesi, Indonesia
[2] Department of Clinical Nutrition, Dr. Wahidin Sudirohusodo General Hospital, Makassar, South Sulawesi, Indonesia
[3]Department of Internal Medicine, Faculty of Medicine, Hasanuddin University, Makassar, South Sulawesi, Indonesia

ABSTRACT: Diabetic kidney disease (DKD) is frequently accompanied by nutritional and inflammatory abnormalities that may not be reflected by glycated hemoglobin (HbA1c) alone. We evaluated immunonutritional status according to HbA1c category and examined whether HbA1c was independently associated with the Prognostic Nutritional Index (PNI) in adults with DKD. This cross-sectional study included 50 patients with type 2 diabetes and DKD. PNI was calculated from serum albumin and total lymphocyte count. HbA1c was analyzed continuously and as <7% versus ≥7%. Group comparisons used the Mann–Whitney U test, independent-samples t-test, or Fisher's exact test as appropriate. Multivariable linear regression assessed the association between HbA1c and PNI after adjustment for eGFR, age, sex, and diabetes duration. Overall, 82.0% of participants had HbA1c <7%, 80.0% had PNI ≤50, 50.0% had PNI ≤40, and 60.0% had hypoalbuminemia; 76.0% had G4–G5 CKD. Among participants with HbA1c <7%, 82.9% had PNI ≤50 and 65.9% had hypoalbuminemia. PNI did not differ significantly between HbA1c groups (39.61 [34.45–48.16] vs 46.34 [36.36–51.36]; p=0.383), nor did serum albumin (3.33±0.72 vs 3.52±0.62 g/dL; p=0.462). HbA1c was not independently associated with PNI (B=−0.298, 95% CI −2.027 to 1.431; p=0.730). Immunonutritional vulnerability was common in this predominantly advanced-DKD cohort, including among patients with HbA1c <7%, supporting nutritional assessment alongside glycemic monitoring.

1. INTRODUCTION

Diabetic kidney disease (DKD) is a major complication of type 2 diabetes and is associated with substantial risks of kidney failure, cardiovascular disease, and premature mortality. Contemporary recommendations therefore emphasize comprehensive management integrating glycemic control with kidney and cardiovascular protection, lifestyle modification, and nutritional care [1,2]. Nutritional impairment is clinically relevant across the chronic kidney disease (CKD) spectrum. Protein-energy wasting has been associated with kidney failure and mortality in nondialysis CKD, while conventional anthropometric measures may incompletely characterize nutritional status, particularly in the presence of altered body composition and fluid imbalance [3-5].

The Prognostic Nutritional Index (PNI), calculated from serum albumin and total lymphocyte count, is a readily available composite marker of immunonutritional status. Among patients with type 2 diabetes and diabetic nephropathy, lower PNI has been associated with subsequent progression to end-stage kidney disease [6]. In broader CKD populations, lower PNI has also

been associated with all-cause and cardiovascular mortality [7-9]. However, PNI should be interpreted as a risk marker rather than a stand-alone diagnostic measure of malnutrition because serum albumin is influenced by inflammation and fluid status, while lymphocyte count reflects both immune and nutritional factors [5].

Glycated hemoglobin (HbA1c) remains central to diabetes monitoring, although its interpretation becomes less straightforward in CKD. CKD-related anemia and carbamylation may modify the relationship between HbA1c and renal outcomes [10], while continuous glucose monitoring studies have demonstrated poor agreement between HbA1c and glucose-derived metrics in patients with CKD and anemia [11]. Less is known about whether conventional HbA1c categories correspond to concurrent immunonutritional status in patients with DKD. Therefore, this study evaluated PNI and serum albumin according to HbA1c category and examined whether HbA1c was independently associated with PNI after accounting for kidney function and key clinical covariates.

2. MATERIALS AND METHODS

2.1 Study design and participants

This study was a focused analysis of data from an observational cross-sectional study conducted at the Kidney-Hypertension Outpatient Clinic of Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia, from May to June 2026. Consecutive sampling was used during the study period. Eligible participants were adults aged ≥18 years with type 2 diabetes mellitus and diabetic kidney disease (DKD) who agreed to participate and provided written informed consent. DKD status was based on the documented clinical diagnosis in the medical record. Patients with chronic liver disease, acute infection or inflammatory conditions, active malignancy, or incomplete study data were excluded. A total of 50 participants with complete data were included in the present analysis. For this manuscript, the analysis focused on the coexistence and association of glycemic status, represented by glycated hemoglobin (HbA1c), with immunonutritional status assessed using the Prognostic Nutritional Index (PNI) and serum albumin.

2.2 Clinical and laboratory measurements

Demographic and clinical information included age, sex, duration of diabetes mellitus, and kidney function. Body weight and height were measured during the study assessment, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. HbA1c, serum albumin, serum creatinine, hemoglobin, and routine hematologic parameters were obtained through the hospital laboratory according to standard operating procedures. The total lymphocyte count used for PNI calculation was obtained from routine hematologic data. The original study protocol included anthropometric assessment and laboratory measurement of HbA1c, serum albumin, and hematologic parameters.

Estimated glomerular filtration rate (eGFR) was recorded as a continuous variable, and CKD G categories were classified according to KDIGO criteria as G1, ≥90; G2, 60–89; G3a, 45–59; G3b, 30–44; G4, 15–29; and G5, <15 mL/min/1.73 m². For descriptive analyses, advanced CKD was defined as eGFR <30 mL/min/1.73 m² (G4–G5). Urine albumin-to-creatinine ratio (UACR) was analyzed categorically as <30 or ≥30 mg/g.

2.3 Glycemic and immunonutritional definitions

HbA1c was analyzed both continuously and categorically, using <7% and ≥7% as a pragmatic analytical threshold for between-group comparisons. This threshold was selected as a conventional glycemic benchmark to facilitate clinically interpretable comparisons and was not intended to represent an individualized glycemic target for all participants with DKD.

PNI was calculated as: PNI = 10 × serum albumin (g/dL) + 0.005 × total lymphocyte count (cells/mm³).

The continuous PNI value was the principal immunonutritional outcome, with higher values indicating a more favorable immunonutritional profile and lower values indicating greater immunonutritional vulnerability. For descriptive analyses, protocol-defined PNI thresholds were converted into mutually exclusive categories: normal (>50), low risk (>45–50), moderate risk (>40–45), and high risk (≤40). PNI ≤50 was additionally used to indicate any immunonutritional risk, whereas PNI ≤40 represented high risk. These categories were used descriptively and should not be interpreted as universally validated diagnostic cutoffs for malnutrition in CKD. Serum albumin was analyzed continuously, and hypoalbuminemia was defined as serum albumin <3.5 g/dL.

2.4 Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 30 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro–Wilk test and inspection of distributional plots. Normally distributed variables are presented as mean ± standard deviation, whereas non-normally distributed variables are presented as median with first and third quartiles (Q1–Q3). Categorical variables are reported as number and percentage.

Because PNI was non-normally distributed within at least one HbA1c category, PNI distributions between participants with HbA1c <7% and ≥7% were compared using the Mann–Whitney U test; the exact two-sided p value was reported. Serum albumin was approximately normally distributed within both HbA1c groups and was compared using an independent-samples t-test after assessment of homogeneity of variance. Differences in the proportions of PNI ≤50, PNI ≤40, and hypoalbuminemia between HbA1c groups were evaluated using two-sided Fisher's exact tests because of small expected cell counts. These were the inferential tests used for the between-group analyses reported in Table 2.

A parsimonious multivariable linear regression model was fitted with continuous PNI as the dependent variable and continuous HbA1c as the main independent variable. The model was adjusted for eGFR, age, sex, and duration of diabetes mellitus, selected as clinically relevant potential confounders while limiting model complexity given the sample size. All variables were entered simultaneously; no automated variable-selection procedure was used. Results are reported as unstandardized regression coefficients (B) with 95% confidence intervals. Model assumptions were assessed using standardized residuals, residual normality testing and plots, residual-versus-predicted plots, and collinearity diagnostics using tolerance and variance inflation factors. Standardized residuals exceeding an absolute value of 3 were considered potentially influential observations. All statistical tests were two-sided, and $p<0.05$ was considered statistically significant.

3. RESULTS AND DISCUSSION

3.1 Participant characteristics

A total of 50 patients with diabetic kidney disease were included, with complete data for all variables analyzed. The mean age was 54.80 ± 10.66 years, and 25 participants (50.0%) were male. Median duration of diabetes was 4.50 years (Q1–Q3, 2.00–8.50), while median body mass index was 17.92 kg/m² (17.33–19.35). Median hemoglobin was 9.30 g/dL (8.30–10.53), and mean total lymphocyte count was 1572.21 ± 664.93 cells/mm³.

Renal impairment was advanced in most participants. Median eGFR was 22.50 mL/min/1.73 m² (14.00–28.75), with 38 participants (76.0%) classified as stage G4–G5 CKD. UACR was ≥30 mg/g in 48 participants (96.0%). Baseline characteristics are summarized in Table 1.

Table 1. Clinical, renal, glycemic, and immunonutritional characteristics of participants (n=50)

Characteristic	Values
Age, years	54.80 ± 10.66
Male sex	25 (50.0)
Duration of diabetes mellitus, years	4.50 (2.00–8.50)
Body mass index, kg/m²	17.92 (17.33–19.35)
Hemoglobin, g/dL	9.30 (8.30–10.53)
Total lymphocyte count, cells/mm³	1572.21 ± 664.93
HbA1c, %	6.04 (5.58–6.72)
HbA1c <7%	41 (82.0)
Serum albumin, g/dL	3.36 ± 0.70
Hypoalbuminemia (<3.5 g/dL)	30 (60.0)

Prognostic Nutritional Index	41.50 ± 8.63
PNI category	
Normal (>50)	10 (20.0)
Low risk (>45–50)	6 (12.0)
Moderate risk (>40–45)	9 (18.0)
High risk (≤40)	25 (50.0)
Serum creatinine, mg/dL	2.85 (2.24–3.52)
eGFR, mL/min/1.73 m²	22.50 (14.00–28.75)
CKD stage	
G1	1 (2.0)
G3a	5 (10.0)
G3b	6 (12.0)
G4	25 (50.0)
G5	13 (26.0)
UACR category	
UACR <30 mg/g	2 (4.0)
UACR ≥30 mg/g	48 (96.0)

Note: Data are presented as mean ± standard deviation, median (Q1–Q3), or n (%), as appropriate. CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; PNI, Prognostic Nutritional Index; Q1, first quartile; Q3, third quartile; UACR, urine albumin-to-creatinine ratio.

3.2 Glycemic and immunonutritional profile

Median HbA1c was 6.04% (5.58–6.72), and 41 participants (82.0%) had HbA1c <7%. In contrast, immunonutritional abnormalities were frequent: 30 participants (60.0%) had hypoalbuminemia, 40 (80.0%) had PNI ≤50, and 25 (50.0%) had PNI ≤40. Thus, only 10 participants (20.0%) were classified as having normal PNI (>50).

3.3 Immunonutritional profile according to HbA1c category

Among participants with HbA1c <7%, 34 of 41 (82.9%) had PNI ≤50, 22 (53.7%) had PNI ≤40, and 27 (65.9%) had hypoalbuminemia. The corresponding proportions among participants with HbA1c ≥7% were 66.7%, 33.3%, and 33.3%, respectively. None of these categorical comparisons reached statistical significance.

Median PNI was 39.61 (34.45–48.16) among participants with HbA1c <7% and 46.34 (36.36–51.36) among those with HbA1c ≥7% (Mann–Whitney U=149.0; exact two-sided p=0.383). Mean serum albumin was also similar between groups (3.33 ± 0.72 vs 3.52 ± 0.62 g/dL; p=0.462) (Table 2).

Table 2. Immunonutritional profile according to HbA1c category

Outcome	HbA1c <7% (n=41)	HbA1c ≥7% (n=9)	p value
Prognostic Nutritional Index	39.61 (34.45–48.16)	46.34 (36.36–51.36)	0.383[a]
Serum albumin, g/dL	3.33 ± 0.72	3.52 ± 0.62	0.462[b]
PNI ≤50	34 (82.9)	6 (66.7)	0.358[c]

PNI ≤40	22 (53.7)	3 (33.3)	0.463[c]
Hypoalbuminemia (<3.5 g/dL)	27 (65.9)	3 (33.3)	0.130[c]

Note: Data are presented as median (Q1–Q3), mean ± standard deviation, or n (%), as appropriate. [a]Mann–Whitney U test, exact two-sided p value. [b]Independent-samples t-test. [c]Fisher exact test, two-sided. PNI, Prognostic Nutritional Index.

The distribution of PNI according to HbA1c category is shown in Figure 1. Although the median PNI was numerically lower among participants with HbA1c <7%, the between-group difference was not statistically significant.

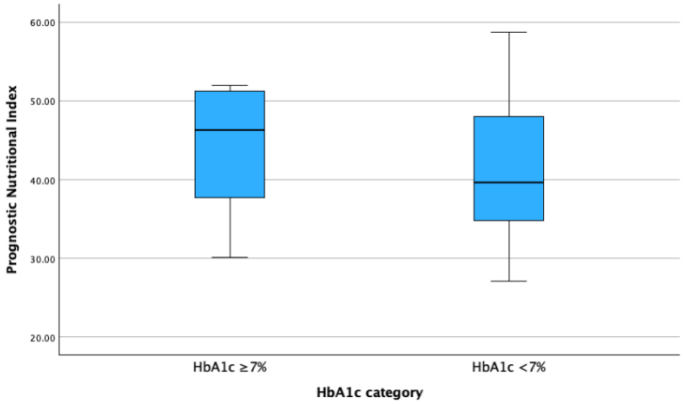

Figure 1. Distribution of Prognostic Nutritional Index according to HbA1c category.

Boxes represent the interquartile range, horizontal lines indicate medians, and whiskers represent the distribution according to the boxplot rule. PNI distributions did not differ significantly between participants with HbA1c <7% and those with HbA1c ≥7% (Mann–Whitney U=149.0; exact two-sided p=0.383).

3.4 Multivariable analysis

In multivariable linear regression, HbA1c was not independently associated with PNI after adjustment for eGFR, age, sex, and diabetes duration (B=−0.298; 95% CI, −2.027 to 1.431; p=0.730). eGFR was also not independently associated with PNI (B=0.107; 95% CI, −0.055 to 0.268; p=0.191), and none of the remaining covariates reached statistical significance. The overall model was not significant (R^2=0.089; adjusted R^2=−0.015; F[5,44]=0.856; p=0.518) (Table 3).

Table 3. Multivariable linear regression of factors associated with Prognostic Nutritional Index

Predictor	B	Standardized β	95% CI for B	p value
HbA1c, per 1% increase	−0.298	−0.055	−2.027 to 1.431	0.730
eGFR, per 1 mL/min/1.73 m² increase	0.107	0.220	−0.055 to 0.268	0.191
Age, per year	−0.027	−0.033	−0.289 to 0.235	0.838
Female vs male	−0.038	−0.002	−5.527 to 5.450	0.989
Duration of diabetes mellitus, per year	0.360	0.172	−0.358 to 1.078	0.318

Note: B represents the unstandardized regression coefficient. All predictors were entered simultaneously. Model R^2=0.089; adjusted R^2=−0.015; F(5,44)=0.856; p=0.518. No problematic multicollinearity was identified (VIF 1.21–1.40). Standardized residuals showed no marked departure from normality (Shapiro–Wilk p=0.083). CI, confidence interval; eGFR, estimated glomerular filtration rate; PNI, Prognostic Nutritional Index; VIF, variance inflation factor.

3.5 Discussion

This study identified a substantial burden of low Prognostic Nutritional Index (PNI) and hypoalbuminemia among patients with diabetic kidney disease (DKD), although most participants had HbA1c <7%. Four in five participants had PNI ≤50, half

had PNI ≤40, and 60% had hypoalbuminemia, while 76% had stage G4–G5 chronic kidney disease (CKD). Malnutrition is common across CKD populations; a meta-analysis of 61 observational studies estimated an overall prevalence of 42.7%, including 38.5% among patients not receiving dialysis [12]. Protein-energy wasting also becomes increasingly relevant in advanced kidney disease, where nutritional abnormalities may reflect reduced intake, catabolic stress, inflammation, and altered body composition rather than body mass index alone [3,13].

The high frequency of low PNI is clinically relevant because contemporary studies have consistently linked this index with adverse kidney and survival outcomes. Lower PNI has been associated with progression of diabetic nephropathy and mortality in populations with diabetes and CKD [6,8,14]. In a cohort of 6,099 adults with CKD, Zhang et al. [7] found that PNI provided the strongest mortality discrimination among several immunonutritional indices and identified a mortality-related threshold of approximately 51. More recently, Gohda et al. [15] reported that low PNI remained associated with kidney events after adjustment for clinical covariates and tumor necrosis factor receptor 2. Their lowest PNI quartile was ≤49.35, although patients with an estimated glomerular filtration rate (eGFR) <30 mL/min/1.73 m² were excluded. These findings support the clinical relevance of PNI values around 50, while also indicating that thresholds are population- and outcome-specific. Accordingly, the PNI ≤40 category used in the present study should be regarded as protocol-defined rather than as a universal CKD cutoff.

A central finding was the frequent coexistence of HbA1c <7% with low PNI or hypoalbuminemia. Among participants with HbA1c <7%, 82.9% had PNI ≤50 and 65.9% had hypoalbuminemia. Nevertheless, neither continuous PNI nor serum albumin differed significantly according to HbA1c category, and HbA1c was not independently associated with PNI after adjustment for eGFR, age, sex, and diabetes duration. A recent longitudinal diabetes cohort likewise showed little difference in HbA1c between participants with lower and higher PNI values (7.3% vs 7.4%), although that population had substantially better baseline kidney function than the present cohort [15]. These observations support interpretation of the present findings as coexistence rather than statistical discordance or biological independence between glycemic and nutritional processes.

Interpretation of HbA1c is particularly important in this cohort because kidney dysfunction was advanced and hemoglobin concentrations were low. CKD-related anemia, altered erythrocyte lifespan, and carbamylation may modify the clinical interpretation of HbA1c [10]. In moderate-to-advanced CKD, continuous glucose monitoring studies have shown that the relationship between HbA1c and glucose-derived indices weakens as kidney function declines, with the weakest agreement observed in stage G5 disease [16]. Reviews and expert consensus similarly indicate that the accuracy and precision of HbA1c decrease in advanced CKD and that continuous glucose monitoring may provide complementary information regarding glucose exposure and variability [17-19].

Accordingly, HbA1c <7% in this study should be considered a pragmatic analytical threshold rather than evidence that every participant had achieved an individualized glycemic target. Current Kidney Disease: Improving Global Outcomes recommendations generally support individualized HbA1c targets ranging from <6.5% to <8.0% among patients with diabetes and CKD not treated with dialysis, depending on comorbidity, hypoglycemia risk, CKD severity, and treatment burden [1,20]. Thus, the clinical implication is not that nutritional vulnerability occurs "despite adequate glycemic control," but rather that a conventional HbA1c threshold does not fully characterize the nutritional and metabolic status of patients with DKD.

The absence of an independent association between eGFR and PNI should also be interpreted cautiously. Nutritional deterioration in CKD is multifactorial. Han et al. [21] found that inadequate energy intake was strongly associated with protein-energy wasting in nondialysis CKD, while low protein intake was associated with protein-energy wasting primarily when energy intake was also inadequate. Serum albumin is additionally influenced by inflammation and fluid status, while lymphocyte count reflects both immune and nutritional processes [5]. Because albumin is itself incorporated into the PNI formula, serum albumin and PNI in the present study should be regarded as overlapping rather than independent measures of immunonutritional status.

From a clinical perspective, these findings support nutritional assessment alongside glycemic monitoring in patients with DKD. Low PNI or hypoalbuminemia may identify individuals who warrant more detailed nutritional evaluation, but neither measure should replace comprehensive assessment incorporating dietary intake, weight trajectory, muscle or body-composition assessment, and validated nutritional tools where appropriate [2,3,5]. Such a multidimensional approach is particularly important in advanced CKD, where fluid imbalance and systemic inflammation may complicate interpretation of isolated anthropometric and biochemical indicators.

This study has several limitations. Its single-center cross-sectional design and modest sample size limit precision, generalizability, and causal inference, while the predominance of stage G4–G5 CKD restricts extrapolation to earlier DKD. HbA1c and nutritional markers were measured at a single time point, continuous glucose monitoring-derived glucose exposure was unavailable, and factors affecting erythrocyte turnover were not comprehensively characterized. PNI cannot distinguish nutritional depletion from inflammation or altered immune status, and its mathematical dependence on serum albumin limits interpretation of PNI and albumin as separate biological outcomes. Finally, no longitudinal kidney or mortality outcomes were available. Prospective studies incorporating repeated nutritional assessment, contemporary glucose monitoring, inflammatory markers, and longitudinal clinical outcomes are therefore needed [18,19].

4. CONCLUSION

Immunonutritional vulnerability was common among patients with diabetic kidney disease, including those with HbA1c <7%. Most participants had PNI ≤50, and hypoalbuminemia was also frequent in this predominantly advanced-CKD population. However, PNI and serum albumin did not differ significantly between HbA1c categories, and HbA1c was not independently associated with PNI after adjustment for eGFR, age, sex, and diabetes duration. These findings suggest that HbA1c alone does not capture concurrent immunonutritional status in DKD. Nutritional assessment should therefore complement glycemic monitoring, particularly in advanced CKD. Prospective studies incorporating repeated nutritional assessment, contemporary glucose monitoring, and longitudinal clinical outcomes are warranted.

ACKNOWLEDGEMENTS

The authors thank all participants who took part in this study. The authors also acknowledge the clinical, laboratory, and research staff of the Kidney-Hypertension Outpatient Clinic, Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia, for their assistance with participant recruitment, clinical assessment, and laboratory measurements. The authors are grateful to the Faculty of Medicine, Hasanuddin University, Makassar, Indonesia, for academic and institutional support.

AUTHOR CONTRIBUTIONS

DVW: Conceptualization, Investigation, Data Curation, Formal Analysis, Visualization, and Writing – Original Draft.

SA: Conceptualization, Methodology, Supervision, Validation, and Writing – Review & Editing.

AYS: Conceptualization, Methodology, Supervision, Validation, and Writing – Review & Editing.

NAD: Methodology, Supervision, Validation, and Writing – Review & Editing.

A: Methodology, Supervision, Validation, and Writing – Review & Editing.

HR: Methodology, Supervision, Validation, and Writing – Review & Editing.

All authors critically reviewed the manuscript, approved the final version, and agreed to be accountable for the integrity of the work.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

FUNDING

This research received no external funding.

ETHICS STATEMENT

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Health Research Ethics Committee of Hasanuddin University, Makassar, Indonesia (Approval No. 555/UN4.6.4.5.31/PP36/2026; Protocol No. UH26030421; April 23, 2026). All participants received information regarding the study procedures and provided written informed consent before enrollment. Participation was voluntary, and participant confidentiality and anonymity were maintained throughout the study.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable ethical and institutional restrictions.

REFERENCES

[1] de Boer, I.H., Khunti, K., Sadusky, T., Tuttle, K.R., Neumiller, J.J., Rhee, C.M., et al., 2022. Diabetes management in chronic kidney disease: A consensus report by the American Diabetes Association (ADA) and Kidney Disease: Improving Global Outcomes (KDIGO). *Diabetes Care* 45(12), 3075–3090.

[2] Stevens, P.E., Ahmed, S.B., Carrero, J.J., Foster, B., Francis, A., Hall, R.K., et al., 2024. KDIGO 2024 Clinical Practice Guideline for the evaluation and management of chronic kidney disease. *Kidney International* 105(4), S117–S314.

[3] Ikizler, T.A., Burrowes, J.D., Byham-Gray, L.D., Campbell, K.L., Carrero, J.J., Chan, W., et al., 2020. KDOQI Clinical Practice Guideline for nutrition in CKD: 2020 update. *American Journal of Kidney Diseases* 76(3), S1–S107.

[4] Franco, B.B., Hopman, W.M., Lamarche, M.C., Holden, R.M., 2022. Protein energy wasting and long-term outcomes in nondialysis dependent chronic kidney disease. *Journal of Renal Care* 48(1), 14–23.

[5] Yang, B., Yang, Y., Liu, B., Yang, M., 2024. Role of composite objective nutritional indexes in patients with chronic kidney disease. *Frontiers in Nutrition* 11, 1349876.

[6] Zhang, J., Xiao, X., Wu, Y., Yang, J., Zou, Y., Zhao, Y., et al., 2022. Prognostic Nutritional Index as a predictor of diabetic nephropathy progression. *Nutrients* 14(17), 3634.

[7] Zhang, J., Xiao, X., Han, T., Liu, Y., Shuai, P., 2023. Relationship between immune nutrition index and all-cause and cause-specific mortality in U.S. adults with chronic kidney disease. *Frontiers in Nutrition* 10, 1264618.

[8] Yu, J.H., Chen, Y., Yin, M.G., 2023. Association between the Prognostic Nutritional Index (PNI) and all-cause mortality in patients with chronic kidney disease. *Renal Failure 45(2)*, 2264393.

[9] Li, W., Chen, L., Jiang, L., Zhang, Z., Song, K., 2025. The Prognostic Nutritional Index as a predictor of cardiovascular and all-cause mortality in chronic kidney disease: A population-based analysis of NHANES data (1999–2018). *Frontiers in Medicine* 12, 1589173.

[10] Tang, M., Berg, A., Rhee, E.P., Allegretti, A.S., Nigwekar, S., Karumanchi, S.A., et al., 2023. The impact of carbamylation and anemia on HbA1c's association with renal outcomes in patients with diabetes and chronic kidney disease. *Diabetes Care* 46(1), 130–137.

[11] Gómez Medina, A., González, C.A., Muñoz, O.M., Gómez, Y., Jaramillo, P.E., Henao, D., et al., 2024. HbA1c overestimates the glucose management indicator: A pilot study in patients with diabetes, chronic kidney disease not on dialysis, and anemia using isCGM. *Therapeutic Advances in Endocrinology and Metabolism* 15, 20420188241252546.

[12] Rashid, I., Bashir, A., Tiwari, P., D'Cruz, S., Jaswal, S., 2021. Estimates of malnutrition associated with chronic kidney disease patients globally and its contrast with India: An evidence based systematic review and meta-analysis. *Clinical Epidemiology and Global Health* 12, 100855.

[13] Narasaki, Y., Rhee, C.M., Kalantar-Zadeh, K., Rastegar, M., 2025. Why protein-energy wasting leads to faster progression of chronic kidney disease. *Current Opinion in Nephrology and Hypertension* 34(1), 55–66.

[14] Zhang, J., Chen, Y., Zou, L., Gong, R., 2023. Prognostic Nutritional Index as a risk factor for diabetic kidney disease and mortality in patients with type 2 diabetes mellitus. *Acta Diabetologica* 60(2), 235-245.

[15] Gohda, T., Kamei, N., Tanaka, M., Furuhashi, M., Sato, T., Kubota, M., et al., 2025. Independent predictive role of nutritional markers in kidney function decline and mortality in diabetes. *Metabolism Open* 27, 100386.

[16] Ling, J., Ng, J.K.C.C., Lau, E.S.H., Ma, R.C.W., Kong, A.P.S., Luk, A.O.Y., et al., 2022. Continuous glucose monitoring metrics in the assessment of glycemia in moderate-to-advanced CKD in diabetes. *Kidney International Reports* 7(6), 1354–1363.

[17] Galindo, R.J., de Boer, I.H., Neumiller, J.J., Tuttle, K.R., 2023. Continuous glucose monitoring to optimize management of diabetes in patients with advanced CKD. *Clinical Journal of the American Society of Nephrology* 18(1), 130–145.

[18] Galindo, R.J., Soliman, D., Cherñavvsky, D., Rhee, C.M., 2024. Diabetes technology in people with diabetes and advanced chronic kidney disease. *Diabetologia* 67(10), 2129–2142.

[19] Rhee, C.M., Gianchandani, R.Y., Kerr, D., Philis-Tsimikas, A., Kovesdy, C.P., Stanton, R.C., et al., 2025. Consensus report on the use of continuous glucose monitoring in chronic kidney disease and diabetes. *Journal of Diabetes Science and Technology* 19(1), 217–245.

[20] Rossing, P., Caramori, M.L., Chan, J.C.N., Heerspink, H.J.L., Hurst, C., Khunti, K., et al., 2022. KDIGO 2022 Clinical Practice Guideline for diabetes management in chronic kidney disease. *Kidney International* 102(5), S1–S127.

[21] Han, Q., Zhang, R., Wu, J., He, F., Qing, F., Li, W., et al., 2024. Cross-sectional relationship between dietary protein intake, energy intake and protein energy wasting in chronic kidney disease patients. *British Journal of Nutrition* 132(3), 309–314.