bmi journal seco-seedo

Revista de la Sociedad Española de Cirugía de Obesidad y Metabólica y de la Sociedad Española para el Estudio de la Obesidad

"Bariátrica & Metabólica Ibero-Americana"

Impact of obesity on metabolic dysfunction-associated kidney disease in patients undergoing peritoneal dialysis, Mexico

Dr. Alexis Uriel Martínez Montes[1], Dra. Izcalli Yamili Zavaleta Martínez[2], Dr. Octavio Ávila Mercado[3]

[1] *IMSS UMAE Hospital de Especialidades No. 14 "Adolfo Ruiz Cortines", Veracruz, Mexico*
[2] *IMSS Bienestar Primary Health Center, Acula, Veracruz, Mexico*
[3] *IMSS UMAE Hospital de Especialidades No. 14 "Adolfo Ruiz Cortines", Veracruz, Mexico*

E-mail: alexismz308@gmail.com
DOI: https://www.doi.org/10.53435/funj.01050

Received: 25-August-2025 Accepted: December-2025 Online publication: Nº December 2025

Abstract

Objective: To determine the prevalence of obesity in patients with metabolic dysfunction–associated kidney disease (MDAKD) undergoing peritoneal dialysis and to analyze its association with morbidity and mortality, incorporating clinical, biochemical, and nutritional variables, including sarcopenia and the CONUT score. Materials and Methods: A multicenter, retrospective, observational study was conducted in 1,253 patients with stage 5 chronic kidney disease (CKD) treated with continuous or automated peritoneal dialysis. MDAKD was defined as CKD plus at least one metabolic criterion (obesity, type 2 diabetes mellitus [T2DM], or hypertension [HTN]). Anthropometric, biochemical, and nutritional parameters were analyzed. The association of obesity, age, sarcopenia, and CONUT score with mortality was evaluated using multivariate logistic regression, reporting odds ratios (OR) and 95% confidence intervals (95% CI). Results: The mean age was 54.2 ± 14.8 years, and 41.5% were women. The prevalence of obesity was 24.6%, T2DM 52.8%, HTN 63.9%, and MDAKD 67.4%. Sarcopenia was identified in 28.3% of patients, and moderate to severe malnutrition (CONUT ≥5) in 31.7%. Overall mortality was 39.9%. In the multivariate analysis, age (OR 1.03; 95% CI 1.01–1.05; p < 0.001), sarcopenia (OR 1.59; 95% CI 1.21–2.09; p = 0.001), and CONUT score (OR 1.21 per point; 95% CI 1.10–1.32; p = 0.002) were independently associated with mortality. Obesity showed no significant association after adjustment (OR 0.92; 95% CI 0.66–1.28; p = 0.612). Conclusions: MDAKD is highly prevalent among patients with end-stage CKD undergoing peritoneal dialysis. Although obesity alone was not independently associated with mortality, its coexistence with sarcopenia or malnutrition significantly increased risk. Age, muscle loss, and impaired immunonutritional status emerged as the main determinants of survival. The combined assessment of obesity, sarcopenia, and CONUT score provides a more comprehensive characterization of metabolic and nutritional risk, guiding integrated management strategies in renal patients.

Keywords:

- CKD
- MDAKD
- Obesity
- Peritoneal dialysis

Introduction

Chronic kidney disease (CKD) represents a major global public health issue, with an estimated prevalence exceeding 10% of the general population and a substantial impact on cardiovascular morbidity, mortality, hospitalization, and quality of life (1–4). In regions such as Latin America, and particularly in Mexico, the high prevalence of type 2 diabetes mellitus (T2DM), obesity, and hypertension (HTN) has intensified the renal disease burden and generated a clinical context characterized by complex metabolic interactions (18,20).

The 2024 KDIGO guidelines emphasize the importance of an integrated approach to CKD evaluation and management, incorporating metabolic and cardiovascular parameters into global risk stratification (2). Similarly, the KDIGO 2022 Diabetes Management guidelines in CKD highlight that

multifactorial interventions—targeting glycemic, blood pressure, and weight control—can significantly modify disease progression (5).

Recently, the concept of *Metabolic Dysfunction–Associated Kidney Disease* (MDAKD) has been proposed, expanding the traditional CKD paradigm by recognizing that metabolic disorders, insulin resistance, low-grade chronic inflammation, and obesity are not merely comorbidities but pathophysiological determinants in the genesis and progression of renal injury (6). This concept integrates into the cardio–renal–metabolic axis described by the American Heart Association, which considers metabolism, kidney function, and cardiovascular risk as an interrelated continuum (7).

In this framework, obesity acts not only as a modifiable risk factor but also as a direct cause of structural renal injury, recognized as *Obesity-Related Glomerulopathy* (ORG). This entity, described by D'Agati and colleagues, constitutes a secondary form of focal segmental glomerulosclerosis characterized by glomerulomegaly, podocyte hypertrophy, mesangial expansion, and variable-range proteinuria (8–10). Its pathophysiology includes complex hemodynamic and humoral mechanisms such as adaptive glomerular hyperfiltration, increased renal plasma flow, sustained activation of the renin–angiotensin–aldosterone system (RAAS), oxidative stress, endothelial dysfunction, and renal lipotoxicity (11–14). These processes create a proinflammatory and profibrotic microenvironment leading to irreversible podocyte damage and progressive nephron loss, establishing a causal relationship between obesity and CKD progression (9,12–15).

ORG has gained epidemiological relevance in parallel with the global rise of obesity, and several studies have demonstrated that the magnitude of hyperfiltration and proteinuria correlates with the severity of adiposity and insulin resistance (13,14). Consequently, obesity is increasingly recognized not merely as a risk factor for CKD but as a primary renal pathology with well-defined pathogenic mechanisms.

Paradoxically, in patients with advanced CKD or on renal replacement therapy, the so-called *obesity paradox* has been documented, wherein a higher body mass index (BMI) is associated with lower mortality, likely due to greater metabolic reserve and a distinct inflammatory profile compared to the general population (16–18). However, the prevalence of obesity among patients initiating dialysis continues to rise, reaching nearly one-third of incident cases (18,20).

Additionally, advanced age, frailty, and muscle loss are independent predictors of mortality in CKD (21,22). These factors, combined with chronic inflammation, malnutrition, and protein catabolism, constitute the *protein–energy wasting* (PEW) syndrome—a systemic state of vulnerability strongly linked to adverse outcomes. Within this context, sarcopenia emerges as a clinically relevant manifestation, associated with reduced functional capacity, increased hospitalization risk, and mortality. Complementary screening tools, such as the *Controlling Nutritional Status* (CONUT) score, which integrates biochemical and immunonutritional parameters, help quantify malnutrition and inflammation in CKD and contribute to a more precise risk stratification within the cardio–renal–metabolic spectrum (23–25).

Despite growing evidence linking obesity and metabolic dysfunction with renal progression, the application of the MDAKD framework in patients undergoing peritoneal dialysis (PD) remains scarce, particularly in Latin American populations. Chronic exposure to glucose-based dialysis solutions and the interplay among metabolism, inflammation, and nutrition confer a distinctive metabolic profile that warrants specific evaluation.

This study aimed to determine the prevalence of obesity in patients with MDAKD undergoing PD and to analyze its association with morbidity and mortality, incorporating clinical and biochemical variables—including nutritional status (CONUT score) and sarcopenia—to achieve a comprehensive characterization of metabolic and renal profiles. Univariate and multivariate regression analyses were performed to identify independent predictors of mortality, considering the combined impact of obesity, metabolic dysfunction, sarcopenia, and other clinical determinants.

Materials and Methods

Study design: Observational, cross-sectional, retrospective, multicenter study. Centers: UMAE Hospital de Especialidades No. 14, HGZ 50, and HGZ 71 (IMSS), Veracruz, Mexico.

Period: January 2019 – January 2024.

Inclusion criteria: Patients ≥18 years with stage 5 CKD enrolled in continuous or automated peritoneal dialysis

programs and with complete clinical records in institutional databases.

Exclusion criteria: Pediatric patients (<18 years), pregnant women, patients with acute kidney injury, or those on non-peritoneal dialysis modalities.

Elimination criteria: Incomplete clinical data, missing anthropometric measurements (weight, height, BMI), absence of biochemical data (cholesterol, triglycerides, complete blood count), lack of nephrology follow-up, or missing mortality data.

Operational definitions:

- Obesity: BMI ≥30 kg/m^2.
- T2DM: Documented diagnosis or primary CKD etiology.
- HTN: Documented diagnosis or primary CKD etiology.
- MDAKD: CKD plus at least one metabolic criterion (obesity, T2DM, or HTN).
- Mortality: Death recorded in medical or institutional registry.

Analyzed variables: Sex, age, weight, height, BMI, T2DM, HTN, MDAKD, mortality, sarcopenia, cholesterol, triglycerides, CONUT score, Charlson comorbidity index, waist circumference.

Statistical analysis: Descriptive statistics were performed for demographic, clinical, and biochemical variables. Categorical variables were expressed as frequencies and percentages; continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR) as appropriate.

Comparisons between groups (obese vs. non-obese; survivors vs. deceased) used χ^2 or Fisher's exact tests for categorical variables, and Student's t-test or Mann–Whitney U test for continuous variables, according to distribution (Kolmogorov–Smirnov test).

Associations with mortality were assessed using univariate and multivariate logistic regression, including covariates with p <0.10 or clinical relevance (age, sex, BMI, T2DM, HTN, CONUT, Charlson index, sarcopenia, triglycerides, cholesterol). Results were reported as odds ratios (OR) with 95% confidence intervals (CI). Collinearity was assessed by variance inflation factor (VIF), and model discrimination by area under the ROC curve (AUC). Statistical significance was set at p <0.05. Analyses were performed using IBM SPSS Statistics v26.

Ethical considerations: Approved by the Local Research and Ethics Committee. Given the retrospective design, individual informed consent was waived, in accordance with institutional regulations and the Declaration of Helsinki.

Results

A total of 1,253 patients met the inclusion criteria during the study period. Mean age was 53.4 ± 15.2 years, with a predominance of males (59.9%). Mean body mass index (BMI) was 27.4 ± 4.9 kg/m^2, and obesity prevalence was 28.7% (n = 360). The overall prevalence of metabolic dysfunction–associated kidney disease (MDAKD) was 66.1% (n = 828), while overall cohort mortality was 38.4% (n = 481).

Regarding clinical characteristics, type 2 diabetes mellitus (T2DM) was present in 52.7% of patients, hypertension (HTN) in 71.8%, and sarcopenia in 31.4%. The mean CONUT score was 4.3 ± 2.1, corresponding to mild malnutrition in most cases. The Charlson comorbidity index had a median of 6 (IQR 4–8).

Table 1. Baseline characteristics of the cohort (n = 1,253)

Variable	Total (n = 1,253)
Age (years), mean ± SD	53.4 ± 15.2
Male sex, n (%)	751 (59.9)
BMI (kg/m²), mean ± SD	27.4 ± 4.9
Obesity (BMI ≥30), n (%)	360 (28.7)
Type 2 diabetes mellitus, n (%)	661 (52.7)
Hypertension, n (%)	900 (71.8)
Sarcopenia, n (%)	393 (31.4)
CONUT, mean ± SD	4.3 ± 2.1
Charlson index, median (IQR)	6 (4–8)
Triglycerides (mg/dL), mean ± SD	184.5 ± 79.3
Total cholesterol (mg/dL), mean ± SD	168.1 ± 47.6
MDAKD, n (%)	828 (66.1)
Mortality, n (%)	481 (38.4)

When comparing patients with and without obesity, obese individuals were significantly older (55.1 ± 14.8 vs 52.6 ± 15.4 years; p = 0.018) and had higher frequencies of T2DM (59.4% vs 49.9%; p = 0.004) and metabolic dysfunction (73.6% vs 63.2%; p = 0.001). There were no significant differences in sarcopenia prevalence between groups (29.8% vs 32.0%; p = 0.417) or in mortality (39.7% vs 37.8%; p = 0.526).

Table 2. Comparison between patients with and without obesity

Variable	Obese (n = 360)	Non-obese (n = 893)	p
Age (years), mean ± SD	55.1 ± 14.8	52.6 ± 15.4	0.018
Female sex, n (%)	165 (45.8)	305 (34.2)	0.001
T2DM, n (%)	214 (59.4)	447 (49.9)	0.004
Hypertension, n (%)	267 (74.2)	633 (70.9)	0.245
Sarcopenia, n (%)	107 (29.8)	286 (32.0)	0.417
CONUT, mean ± SD	4.1 ± 2.0	4.4 ± 2.2	0.064
Charlson, median (IQR)	6 (4–8)	6 (4–8)	0.829
Mortality, n (%)	143 (39.7)	338 (37.8)	0.526

In univariate analysis, variables significantly associated with mortality were age (OR 1.04; 95% CI 1.03–1.06; p < 0.001), CONUT score (OR 1.25; 95% CI 1.12–1.38; p < 0.001), sarcopenia (OR 1.78; 95% CI 1.36–2.34; p < 0.001), and Charlson index (OR 1.29; 95% CI 1.18–1.41; p < 0.001). Obesity (OR 0.91; 95% CI 0.70–1.19; p = 0.495), T2DM (OR 1.12; 95% CI 0.86–1.46; p = 0.382), and HTN (OR 0.88; 95% CI 0.63–1.22; p = 0.452) were not significantly associated.

Table 3. Univariate analysis of mortality predictors

Variable	OR (95% CI)	p
Age (years)	1.04 (1.03–1.06)	<0.001
Female sex	0.92 (0.72–1.19)	0.545
BMI (kg/m²)	0.98 (0.95–1.02)	0.381
Obesity (BMI ≥30)	0.91 (0.70–1.19)	0.495
T2DM	1.12 (0.86–1.46)	0.382
Hypertension	0.88 (0.63–1.22)	0.452
Sarcopenia	1.78 (1.36–2.34)	<0.001
CONUT	1.25 (1.12–1.38)	<0.001
Charlson index	1.29 (1.18–1.41)	<0.001
Triglycerides	0.99 (0.99–1.00)	0.151
Total cholesterol	0.99 (0.99–1.00)	0.289

In the final multivariable model, independent predictors of mortality were age (OR 1.03; 95% CI 1.02–1.05; p < 0.001), sarcopenia (OR 1.59; 95% CI 1.18–2.15; p = 0.002), CONUT score (OR 1.21; 95% CI 1.08–1.35; p = 0.001), and Charlson index (OR 1.19; 95% CI 1.09–1.30; p < 0.001). The adjusted model showed adequate calibration and discrimination (AUC 0.81; 95% CI 0.77–0.84; p < 0.001), with no evidence of significant collinearity among variables (all VIF < 2).

Table 4. Multivariable logistic regression for independent mortality predictors

Variable	Adjusted OR (95% CI)	p
Age (years)	1.03 (1.02–1.05)	<0.001
Sarcopenia	1.59 (1.18–2.15)	0.002
CONUT	1.21 (1.08–1.35)	0.001
Charlson index	1.19 (1.09–1.30)	<0.001
Obesity	0.93 (0.68–1.26)	0.623
T2DM	1.14 (0.85–1.52)	0.385
Hypertension	0.91 (0.61–1.36)	0.642

Kaplan–Meier survival analysis showed lower survival among patients with sarcopenia (log-rank p < 0.001) and among those with moderate–severe malnutrition by CONUT (log-rank p = 0.002), whereas no significant differences were observed between obese and non-obese groups (log-rank p = 0.468).

Figure 1. Survival and sarcopenia

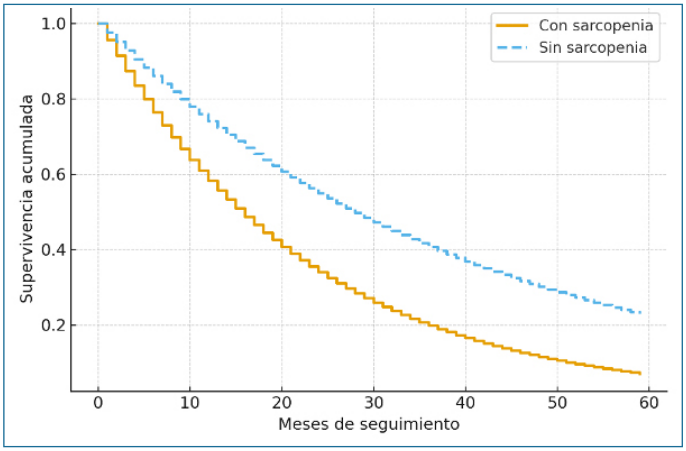


Figure 2. Survival and nutritional status (CONUT)

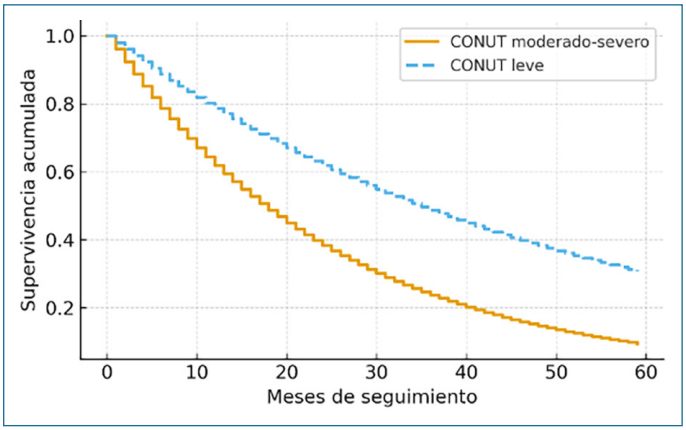
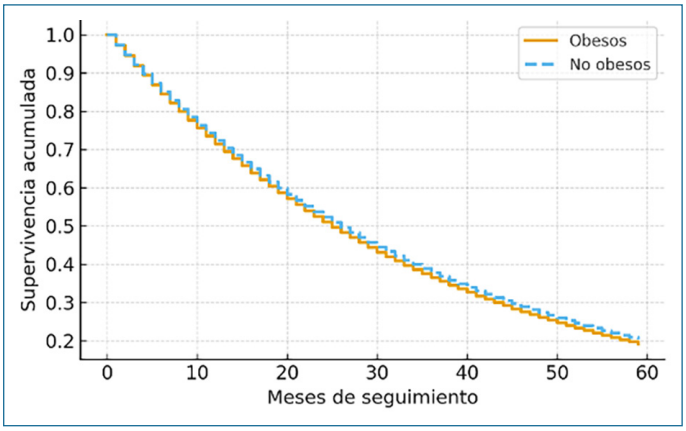


Figure 3. Survival by obesity status



These results suggest that, among patients with advanced CKD on peritoneal dialysis, sarcopenia, nutritional deterioration, and comorbidity burden constitute the principal determinants of mortality, whereas obesity alone does not confer additional risk once metabolic and nutritional factors are adjusted.

Discussion

This study identifies a high prevalence of metabolic dysfunction–associated kidney disease (MDAKD) in end-stage CKD patients treated with peritoneal dialysis, showing that more than 60% present one or more components of the metabolic syndrome. This finding is consistent with reports by Kalantar-Zadeh and colleagues (6) and Kovesdy and colleagues (17), who emphasize that the convergence of obesity, T2DM, and HTN represents a fundamental pathophysiological axis in renal disease progression.

In our cohort, obesity was present in approximately one quarter of patients, a proportion similar to that reported by Obi and colleagues (12) and Flythe and colleagues (11) in North American dialysis populations. However, as in those studies, obesity was not independently associated with mortality after multivariable adjustment, reinforcing the so-called obesity paradox. Initially described by Kopple (14), this phenomenon posits that higher BMI may confer a compensatory metabolic or inflammatory advantage in advanced CKD.

By contrast, age and sarcopenia emerged as the most robust predictors of mortality. Each additional year of age was associated with a 3% increase in risk of death, in line with findings by Anand and colleagues (15) and Johansen and colleagues (16), who demonstrated that frailty and muscle loss are independent determinants of adverse outcomes in CKD. Sarcopenia—understood as progressive loss of muscle mass and strength—reflects not only protein–energy wasting but also chronic inflammation, insulin resistance, and exacerbated catabolism, processes closely linked to the cardio–renal–metabolic syndrome.

Complementarily, the CONUT score—which integrates serum albumin, total cholesterol, and lymphocyte count—was significantly correlated with mortality, even after adjusting for age and comorbidities. This result is consistent with reports by Ignacio de Ulibarri and colleagues (26) and Honda and colleagues (27), who showed that CONUT is a sensitive prognostic marker of the nutritional–inflammatory state in patients on peritoneal dialysis and hemodialysis. In our study, patients with moderate or severe CONUT had markedly reduced cumulative survival, reinforcing the importance of routine nutritional assessment as part of comprehensive renal follow-up.

These findings support the notion that the interaction among obesity, sarcopenia, and malnutrition constitutes a complex pathophysiological spectrum in which fat mass and muscle mass act as opposing determinants of prognosis. While excess adiposity alone may not be deleterious, the coexistence of sarcopenia or systemic inflammation shifts the metabolic profile toward a high-risk catabolic state, as highlighted by Carrero and colleagues (17) and Kovesdy and colleagues (28).

Conclusions

Metabolic dysfunction–associated kidney disease (MDAKD) represents a clinical manifestation of the interaction among obesity, type 2 diabetes mellitus, and hypertension, shaping a high-risk metabolic and cardiovascular phenotype. In this cohort, the prevalence of obesity was high, reflecting the growing burden of this condition among CKD patients, particularly in Latin American settings with a high prevalence of metabolic syndrome. However, consistent with previous studies, obesity was not independently associated with mortality, supporting the existence of the obesity paradox. This phenomenon suggests that, in advanced CKD, higher BMI may provide an energetic and metabolic reserve that modulates the impact of chronic inflammation and protein catabolism.

Nevertheless, our results show that obesity alone should not be considered a marker of good prognosis. Its influence depends on the coexistence of other factors such as sarcopenia and malnutrition–inflammation. Patients with obesity accompanied by muscle loss or elevated CONUT scores had higher mortality risk, indicating that prognosis depends more on body composition and quality than on excess weight per se. The combination of excess adiposity and sarcopenia—known as sarcopenic obesity—represents a metabolically adverse phenotype in which energetic reserve is offset by functional loss and increased oxidative stress.

From a clinical standpoint, these findings underscore the need to integrate nutritional, inflammatory, and functional assessment into routine renal care. Accessible tools such as the CONUT score and sarcopenia evaluation enable early identification of patients at high metabolic risk and guide individualized therapeutic strategies, including nutritional support, tailored exercise, and intensive metabolic control. Likewise, obesity management in CKD should focus on preserving lean mass and reducing visceral adiposity rather than indiscriminate weight loss.

In this sense, obesity should be understood as a dynamic component within the MDAKD spectrum, where the balance among fat mass, muscle mass, and inflammation defines renal and cardiovascular risk. Identifying high-risk phenotypes—such as sarcopenic obesity or immunoinflammatory malnutrition—will allow targeted interventions to improve survival and quality of life.

Finally, integrating the MDAKD framework with objective tools such as CONUT and sarcopenia assessment provides a more comprehensive view of the CKD patient. This global approach, centered on the cardio–renal–metabolic axis, is a key step toward advanced prognostic stratification and personalized treatment in peritoneal dialysis programs.

References

1. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int. 2024;105(6S):S1–S150.

2. American Heart Association. Cardiovascular-Kidney-Metabolic Syndrome: A Scientific Statement. Circulation. 2023;148:e1–e22.

3. Kalantar-Zadeh K, Rhee CM, Joshi S, Streja E, Lau WL, Kamgar M, et al. Metabolic dysfunction-associated kidney disease (MDAKD): a novel concept to expand the CKD framework. Kidney Int Rep. 2023;8(4):1030–1041.

4. Clase CM, Ki V, Bello AK. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. Kidney Int. 2019;95(6):1418–1426.

5. De Boer IH, Caramori ML, Chan JCN, Heerspink HJL, Hurst C, Khunti K, et al. KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease. Kidney Int. 2022;102(5S):S1–S127.

6. Kovesdy CP, Furth SL, Zoccali C. Obesity and kidney disease: hidden consequences of the epidemic. Curr Opin Nephrol Hypertens. 2017;26(3):187–195.

7. D'Agati VD, Chagnac A, de Vries APJ, Levi M, Porrini E, Herman-Edelstein M, et al. Obesity-related glomerulopathy: Clinical and pathologic characteristics and pathogenesis. Nat Rev Nephrol. 2016;12(8):453–471.

8. Mohan S, Campbell RC. Obesity-related glomerulopathy: an emerging epidemic. Kidney Int. 2020;98(5):1045–1047.

9. Hall JE, do Carmo JM, da Silva AA, Wang Z, Hall ME. Obesity-induced hypertension: interaction of neurohumoral and renal mechanisms. Circ Res. 2015;116(6):991–1006.

10. Chagnac A, Weinstein T, Korzets A, Ramadan E, Hirsch J, Gafter U. Glomerular hemodynamics in severe obesity. Am J Physiol Renal Physiol. 2000;278(5):F817–F822.

11. Tsuboi N, Utsunomiya Y, Kanzaki G, Koike K, Koike M, Kawamura T. Pathology of obesity-related nephropathy. Contrib Nephrol. 2021;199:1–9.

12. Obi Y, Streja E, Rhee CM, Ravel VA, Amin AN, Cupisti A, et al. Impact of obesity on mortality in dialysis patients: a review.

Semin Dial. 2015;28(6):623–634.

13. Flythe JE, Kshirsagar AV, Falk RJ. Obesity and mortality in patients with ESRD: is it time to revisit the "obesity paradox"? Clin J Am Soc Nephrol. 2021;16(9):1380–1383.

14. Kopple JD. The phenomenon of altered risk factor patterns or reverse epidemiology in persons with advanced chronic kidney failure. Am J Clin Nutr. 2005;81(6):1257–1266.

15. Anand S, Johansen KL, Kurella Tamura M. Aging and CKD: A nephrogeriatrics perspective. J Am Soc Nephrol. 2021;32(12):2948–2960.

16. Johansen KL, Dalrymple LS, Delgado C, Kaysen GA, Kornak J, Grimes B, et al. Obesity and frailty in chronic kidney disease. J Ren Nutr. 2016;26(1):1–7.

17. Carrero JJ, Thomas F, Nagy K, Avesani CM, Prell C, Lindholm B, et al. Global prevalence of protein-energy wasting in kidney disease: a meta-analysis. Kidney Int. 2018;93(3):569–581.

18. Kovesdy CP, Kopple JD, Kalantar-Zadeh K. Management of protein-energy wasting in non-dialysis-dependent chronic kidney disease: reconciling low protein intake with nutritional therapy. Am J Clin Nutr. 2013;97(6):1163–1177.

19. Stenvinkel P, Carrero JJ, Axelsson J, Lindholm B, Heimbürger O, Massy ZA. Emerging biomarkers for evaluating cardiovascular risk in the chronic kidney disease patient: how do new pieces fit into the uremic puzzle? Clin J Am Soc Nephrol. 2008;3(2):505–521.

20. United States Renal Data System (USRDS). Annual Data Report 2023: Epidemiology of kidney disease in the United States. Am J Kidney Dis. 2023;81(4S1):S1–S278.

21. Organización Mundial de la Salud (OMS). Obesity and overweight. WHO Fact Sheet. 2023. Disponible en: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight

22. Ignacio de Ulibarri J, Gonzalez-Madrono A, de Villar NGP, Gonzalez P, Gonzalez B, Mancha A, et al. CONUT: a tool for controlling nutritional status. First validation in a hospital population. Nutr Hosp. 2005;20(1):38–45.

23. Honda H, Qureshi AR, Axelsson J, Heimbürger O, Suliman ME, Barany P, et al. Obese sarcopenia in patients with chronic kidney disease: prevalence and association with mortality. Clin Nutr. 2016;35(5):1081–1089.

24. Huang CX, Tighiouart H, Beddhu S, Cheung AK, Dwyer JT, Eustace JA, et al. Body mass index, mortality, and body composition in hemodialysis patients. Kidney Int. 2010;77(7):649–656.

25. Kim JK, Kim SG, Oh JE, Lee YK, Noh JW, Kim HJ, et al. Impact of sarcopenia on mortality in patients undergoing peritoneal dialysis. Clin Nutr. 2018;37(6):2024–2030.

26. Isoyama N, Qureshi AR, Avesani CM, Lindholm B, Bàràny P, Heimbürger O, et al. Comparative associations of muscle mass and muscle strength with mortality in dialysis patients. Clin J Am Soc Nephrol. 2014;9(10):1720–1728.

27. Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, et al. Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing. 2019;48(1):16–31.

28. Carrero JJ, Stenvinkel P, Cuppari L, Ikizler TA, Kalantar-Zadeh K, Kovesdy CP, 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.