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Original Article

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Can Serum Uric Acid Levels Serve as a Marker of Mortality in Peritoneal Dialysis Patients? A Single-Center Experience

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ABSTRACT

Background: Evidence regarding the clinical implications of uric acid (UA) levels in end-stage kidney disease remains inconsistent, reflecting the complexity of its prognostic role. Findings from prior investigations into UA and mortality in dialysis cohorts have been conflicting. Accordingly, this study sought to explore whether longitudinally averaged serum UA levels are associated with all-cause mortality among patients undergoing peritoneal dialysis (PD).
Methods: This retrospective observational cohort study included 50 adult patients who initiated PD between January 1, 2012, and July 31, 2022. Serum UA levels were measured every 3 months from PD initiation until death, transfer to hemodialysis, transplantation, or end of follow-up. Time-averaged UA was calculated as the mean of all measurements per patient. The primary endpoint was all-cause mortality. Predictive accuracy was evaluated through receiver operating characteristic (ROC) curve analysis and survival assessment using the Kaplan–Meier method.
Results: The median age at PD initiation was 52.5 years (interquartile range [IQR]: 37.8-59), and 52% were male. Ten patients (20%) died during follow-up. Median time to death was 570 days (IQR, 193-1691). The ROC analysis identified a time-averaged UA cutoff of 5.75 mg/dL (sensitivity = 80%, specificity = 75%; area under the curve = 0.739, 95% CI = 0.579-0.898, P = .021). Kaplan–Meier survival curves demonstrated a significantly greater mortality risk among patients with UA levels below the identified cutoff (log-rank P = .009).
Conclusion: Lower time-averaged UA levels were associated with increased mortality risk in PD patients. These exploratory findings require validation in larger multicenter studies.
Keywords: End-stage kidney disease, mortality, peritoneal dialysis, uric acid

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INTRODUCTION

Peritoneal dialysis (PD) is a key kidney replacement therapy for end-stage kidney disease (ESKD), offering several advantages over hemodialysis (HD), including better fluid management and better preservation of kidney function, reduced hemodynamic stress, lower infection risk, and improved stability.[1,2,3] Despite these benefits, mortality rates among ESKD patients on PD remain high. In addition to traditional risk factors, non-traditional factors such as inflammation and oxidative stress significantly contribute to these outcomes. Increasing attention has focused on uric acid (UA), a metabolite derived from purine catabolism, because of its complex biological profile that encompasses both protective antioxidative activity and pro-inflammatory effects.[4]

The effect of UA on health outcomes is multifaceted. Studies examining UA levels and mortality in HD patients have yielded heterogeneous results.[5-8] A limited number of studies in PD patients have also revealed inconsistent associations. Some have reported an increased risk of

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death with high serum UA levels, while others have reported a decreased risk. In addition, some authors have reported a U-shaped, bidirectional relationship between UA levels and mortality.[9-11] These conflicting findings highlight the complexity of the role of UA in prognosis. Therefore, instead of evaluating a single spot UA measurement, time-averaged UA values may provide a more accurate assessment of prognosis and help improve health outcomes.

This study aimed to investigate the relationship between serum UA levels and mortality in ESKD patients newly initiated on PD treatment, using time-averaged UA levels instead of a single measurement.

MATERIAL AND METHODS

This retrospective observational cohort study was conducted on patients who began PD treatment at the Nephrology Department of Dışkapı Training and Research Hospital. The study covered the period from January 1, 2012, to July 31, 2022, and received ethics approval from the Local Ethics Committee of the hospital. The research adhered to the principles of the Declaration of Helsinki.

The study included patients aged 18 years and older who initiated PD in the Nephrology Department. Exclusion criteria were PD duration of less than 3 months, history of organ transplantation or hemodialysis before PD initiation, incomplete follow-up data, history of gout, and use of medications potentially affecting serum UA levels, including allopurinol as UA-lowering therapy, losartan because of its uricosuric effect, and furosemide because of its potential effect on serum UA levels. Based on these criteria, the study focused on a total of 50 patients. The primary endpoint was all-cause mortality. Analyses were completed when a patient died, returned to HD, underwent kidney transplantation, or when the study ended on July 31, 2022.

The study utilized retrospective data from PD records and the hospital database, including both laboratory and clinical parameters. Demographic and clinical information, as well as laboratory data at the onset of PD treatment, were recorded. This included age, sex, height, weight, systolic and diastolic blood pressure, the primary cause of kidney failure, and the presence of comorbidities such as diabetes mellitus (DM), hypertension (HT), and atherosclerotic heart disease. Body mass index (BMI) was calculated using the formula: weight in kilograms (kg) divided by height in meters squared (m^2). Baseline values for daily urine output and laboratory parameters—including blood urea, serum creatinine, albumin, UA, fasting blood sugar, C-reactive protein (CRP), calcium, phosphorus, parathyroid hormone, ferritin, lipid profiles, and standard (std) Kt/V—were also collected. Serum UA levels, albumin, weight, std Kt/V, CRP, and urine volumes were recorded every 3 months from the start of PD therapy until death, conversion to HD, transplantation, or completion of the study. Time-averaged UA levels were based on means from measurements taken at 3-month intervals and were calculated using the formula: [(N1 + N2 + N3 + ... + Nn) / n]. Informed consent was waived as the study was retrospective and based on anonymized hospital data.

Statistical Analysis

Analyses were executed using SPSS software, version 24 (IBM SPSS Corp.; Armonk, NY, USA). To determine whether continuous variables followed a normal distribution, the Kolmogorov–Smirnov test was applied. For normally distributed continuous data, comparisons were made using ANOVA, with results reported as mean ± SD. For non-normally distributed data, the Mann-Whitney *U*-test was used, and results were presented as median values with interquartile ranges. Categorical variables were compared using chi-square and Fisher's exact tests, with results reported as counts and percentages. Receiver operating characteristic (ROC) curve analysis determined optimal cutoff values of time-varying UA for predicting mortality, with UA evaluated as both continuous and categorical variables. Kaplan–Meier survival curves and univariable and parsimonious multivariable Cox regression models were used to assess the impact of UA on mortality. Variables with $P < .05$ in the univariable analysis were included in the multivariable model. Logarithmically transformed values were used for non-normally distributed data in the Cox regression analyses. Hazard ratios (HRs) with corresponding 95% CI were used to express associations. A *P*-value less than .05 was accepted as indicating statistical significance.

RESULTS

Fifty patients who initiated PD treatment between 2012 and 2022 were included in this study. No patients were lost to follow-up during the study. All enrolled patients were followed until death, conversion to HD, kidney transplantation, or the end of the observation period. Table 1 summarizes the demographic and clinical characteristics at the onset of PD. The causes of ESKD were DM (n = 12, 24%), HT (n = 41, 82%), polycystic kidney disease (n = 2, 4%), nephrolithiasis (n = 2, 4%), glomerular diseases (n = 5, 10%), and other causes (n = 5, 10%). The

MAIN POINTS

- In peritoneal dialysis (PD) patients, lower time-averaged serum uric acid (UA) levels were independently associated with higher all-cause mortality.
- A time-averaged UA threshold of 5.75 mg/dL predicted mortality with acceptable discriminatory ability (area under the curve = 0.739; sensitivity = 80%, specificity = 75%).
- Patients with UA levels below 5.75 mg/dL had significantly worse survival in Kaplan–Meier analysis.
- Unlike single-point measurements, longitudinal time-averaged UA values may better reflect prognosis in PD patients.
- Low UA levels may reflect as a surrogate marker of adverse nutritional or clinical status rather than a direct pathogenic factor.

Table 1. Demographic, Clinical, and Laboratuary Findings of the Patients

Variables	All Patients, n = 50	Survivors, n = 40	Non-Survivors, n = 10	*P*
Age (years)	52.50 (37.75-59)	48.55 ± 14.38	55.80 ± 18.27	.18
Sex (male), n (%)	26 (52)	20 (40)	6 (12)	.72
Time averaged body mass index (kg/m^2)	26.3 ± 4.2	26.3 ± 4.4	26.4 ± 3.0	.93
Systolic blood pressure (mm/Hg)	130 ± 16	127 ± 14	140 ± 20	.02
Diastolic blood pressure (mmHg)	79 ± 12	78 ± 13	84 ± 21	.11
Time averaged urine output (mL/day)	1500 (925-2000)	1550 (1013-2075)	1500 (1000-1525)	.02
Time averaged std Kt/V	2.61 ± 0.52	2.49 ± 0.39	2.36 ± 0.40	.38
Comorbidities, n (%)				
Hypertension	41 (82)	31 (77.5)	10 (100)	.17
Diabetes mellitus	12 (24)	7 (17.5)	5 (50)	.04
Atherosclerotic heart disease	12 (24)	8 (20)	4 (40)	.22
Laboratuary parameters				
Urea (mmol/L)	179 ± 37	187 ± 38	159 ± 25	.13
Serum creatinine (mg/dL)	7.47 ± 1.65	8.2 ± 2.03	7.18 ± 1.41	.44
Time averaged serum albumin (g/dL)	3.61 ± 0.45	3.66 ± 0.48	3.39 ± 0.3	.03
Uric acid (mg/dL)	7.64 ± 1.51	7.80 ± 1.49	7.01 ± 1.49	.14
Time averaged UA	6.07 ± 0.97	6.25 ± 0.97	5.66 ± 0.86	.08
Fasting blood glucose (mg/dL)	103 ± 30	100 ± 25	116 ± 44	.12
Time averaged C–reactive protein (mg/dL)	7 (3.15-13.92)	7 (3.32-13.97)	6 (2.16-14)	.55
Calcium (mg/dL)	8.29 ± 0.88	8.27 ± 0.88	8.40 ± 0.90	.68
Phosphorus (mg/dL)	5.47 ± 1.49	5.53 ± 1.43	5.24 ± 1.76	.58
Parathyroid hormone (ng/L)	341 (262-535)	339 (266-579)	347 (236-401)	.45
Ferritin (µg/L)	188 (109-310)	199 (108-311)	142 (126-328)	.85
Total cholesterol (mg/dL)	177 (166-215)	176 (166-206)	208 (157-231)	.26
LDL cholesterol (mg/dL)	125 (92-147)	121 ± 36	144 ± 37	.24
Triglyceride (mg/dL)	151 (108-193)	146 (109-197)	157 (106-174)	.33

Data are presented as mean ± standard deviation (SD) or median (interquartile range [IQR]), as appropriate.
LDL, low-density lipoprotein; UA, uric acid.

primary etiology remained unclear in 6 (12%) patients. Among the patients, 82% had HT, 24% had DM, and 24% had atherosclerotic heart disease.

The median age at the start of PD was 52.5 years (range: 37.8-59), with 52% of patients being male. The mean BMI was 26.3 ± 4.2 kg/m^2, and the median urine output was 1500 mL/day (range: 925-2000). No patient was oliguric at PD initiation. Table 1 summarizes the patients' outcomes at the end of the study: 38% remained on PD, 26% switched to HD, 16% underwent kidney transplantation, and 20% died. Table 1 presents the baseline laboratory parameters and time-averaged UA values. No patients had a history of gout or UA-lowering medication use.

The median time to death after the initiation of PD was 570 days (interquartile range [IQR]: 193-1691 days). During the study period, 10 patients (20%) died. The causes of death were as follows: acute myocardial infarction (n = 2), cerebrovascular accident and its complications (n = 2), fungal peritonitis (n = 1), bacterial pneumonia (n = 1), COVID-19 pneumonia (n = 2), and 2 sudden deaths of unknown cause. To evaluate outcome differences, individuals were grouped according to whether death occurred during follow-up (Table 1). Both baseline urine volume and serum albumin levels were significantly lower in the non-survivors (P = .04 and P = .03, respectively), while the prevalence of DM was higher in this group (50% vs. 17.5%, P = .04). The effect of time-averaged UA on mortality was compared using both the linear value and the cutoff value. The cutoff UA value

was determined to be 5.75 mg/dL by ROC analysis (sensitivity = 80%, specificity = 75%) [(area under the curve, AUC = 0.739 (95% CI: 0.579-0.898), *P* = .021)] (Figure 1). The Kaplan–Meier survival curve showed that patients with UA levels below 5.75 mg/dL had a higher risk of mortality (*P* = .009, log-rank test) (Figure 2). Cox regression analyses revealed that time-averaged UA was significantly associated with mortality in both univariate and multivariate analyses, with lower UA levels correlating with an increased risk of mortality (Table 2). An additional Spearman correlation analysis was performed to evaluate the relationship between time-averaged serum UA and time-averaged CRP levels. Time-averaged UA showed no significant correlation with time-averaged CRP (rho = 0.078, *P* = .59).

To evaluate the robustness of the ROC-derived UA threshold, exploratory sensitivity analyses were performed using adjacent alternative cutoff values of 5.5 mg/dL and 6.0 mg/dL, in addition to the primary ROC-derived cutoff of 5.75 mg/dL (Table 3). The ROC-derived 5.75 mg/dL threshold showed the strongest association with mortality in univariate Cox regression analysis (HR: 0.19, 95% CI: 0.04-0.75, *P* = .01). At the 6.0 mg/dL threshold, patients with time-averaged UA levels <6.0 mg/dL had a higher mortality rate than those with UA levels ≥6.0 mg/dL, and Kaplan–Meier analysis demonstrated poorer survival in this group (log-rank *P* = .041; Figure 3). At the 5.5 mg/dL threshold, mortality was also numerically higher among patients with UA levels <5.5 mg/dL; however, the survival difference did not reach statistical significance (log-rank *P* = .208; Figure 4). Overall, these findings suggest that the association between lower UA levels and mortality was most evident around the ROC-derived threshold and the adjacent 6.0 mg/dL cutoff, whereas the 5.5 mg/dL threshold showed a weaker and statistically non-significant pattern.

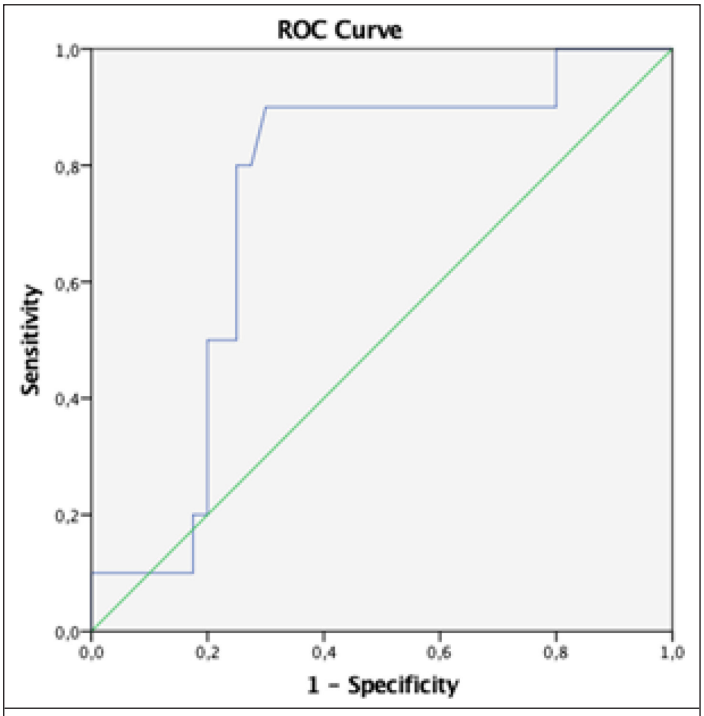

Figure 1. ROC curve showing the predictive ability of time-averaged uric acid levels for all-cause mortality in peritoneal dialysis patients. The blue line represents the ROC curve for time-averaged uric acid. AUC = 0.739 (95% CI: 0.579-0.898), *P* = .021. The optimal cutoff value was determined as 5.75 mg/dL, with a sensitivity of 80% and specificity of 75%. The green diagonal line represents a random classifier (AUC = 0.5). AUC, area under the curve; ROC, receiver operating characteristic curve.

DISCUSSION

This study investigated the relationship between time-averaged UA levels and all-cause mortality in patients undergoing PD. The results showed that lower time-averaged UA levels, based on follow-up measurements after initiating PD, were associated with an increased risk of all-cause mortality. Notably, the risk of mortality increased as time-averaged UA values decreased**,** with an ROC-derived threshold identified at 5.75 mg/dL.

The relationship between UA levels and mortality in dialysis patients remains unclear. Serum UA levels may be linked to mortality risk through complex pathways involving both protective and harmful mechanisms. Elevated UA levels in patients with chronic kidney disease and in the general population have been associated with oxidative stress, inflammation, endothelial dysfunction, and activation of the renin–angiotensin system. These factors may increase the risk of hypertension, peripheral arterial disease, diabetes, kidney disease progression, and atherosclerotic heart disease.[12]

Conversely, UA also serves as a nutritional marker, with hyperuricemia often reflecting better nutritional status.[8] Higher UA levels have been associated with fewer hospitalizations and lower cardiovascular mortality, particularly in HD patients. Furthermore, both in vitro and in vivo studies show that UA

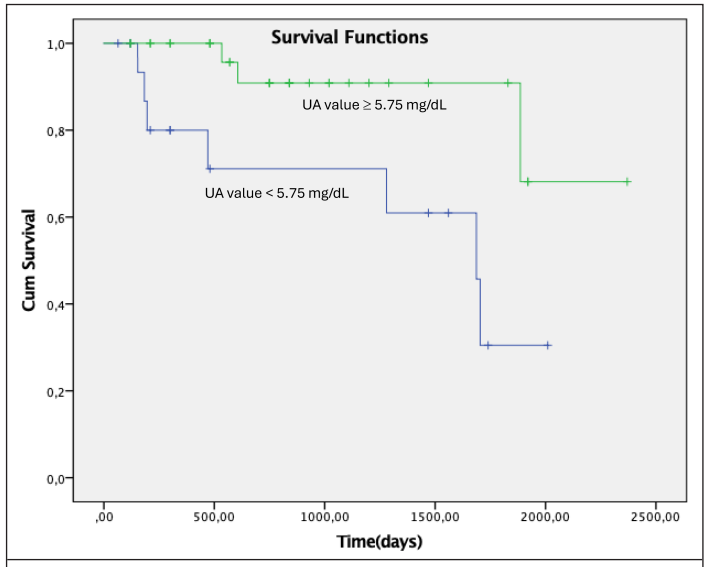

Figure 2. The Kaplan–Meier survival curve showed that patients with UA levels below 5.75 mg/dL had a higher risk of mortality (*P* = .009, log-rank test). UA, uric acid.

Table 2. Evaluation of Risk Factors for Mortality Using Univariate and Parsimonious Multivariate Regression Analysis

	Univariate		Multivariate	
Variables	**HR (95% CI)**	***P***	**HR (95% CI)**	***P***
Age (years)	1.04 (0.99-1.08)	.08		
Sex	0.61 (0.15-2.50)	.5		
Time averaged body mass index	0.99 (0.86-1.15)	.93		
Systolic blood pressure	1.05 (1.01-1.09)	.01	1.026 (0.973-1.083)	.34
Diastolic blood pressure	1.03 (0.99-1.07)	.15		
Time averaged urine output	0.99 (0.98-1.00)	.11		
Diabetes mellitus	9.29 (2.13-40.56)	.03	6.980 (1.59-30.69)	.01
Atherosclerotic heart disease	2.51 (0.70-9.40)	.15		
Fasting blood glucose	1.03 (1.008-1.05)	.007	1.028 (0.99-1.03)	.4
Urea	0.98 (0.97-1.02)	.11		
Creatinine	1.10 (0.84-1.45)	.46		
Time averaged std Kt/V	0.87(0.11-6.92)	.89		
Time averaged albumin	0.07 (0.009-9.57)	.01	0.092 (0.2-0.5)	.01
Time averaged C-reactive protein	0.99 (0.92-1.13)	.76		
Time averaged UA	0.38 (0.18-0.98)	.046	0.301 (0.09-0.94)	.03
Cutoff UA	0.19 (0.04-0.75)	.01	0.234 (0.56-0.986)	.04
Ferritin	1.01 (0.99-1.00)	.98		
Calcium	1.13 (0.59-2.16)	.7		
Phosphorus	0.91 (0.63-1.33)	.64		
Parathyroid hormone	0.99 (0.99-1.01)	.14		
Total cholesterol	1.01 (0.99-1.02)	.5		
LDL cholesterol	1.01 (0.99-1.02)	.22		
Triglyceride	0.99 (0.99-1.00)	.31		

CI, confidence interval; HR, hazard ratio; LDL, low-density lipoprotein; UA, uric acid.

acts as a potent antioxidant and scavenger of oxygen-free radicals.[7,13-16] A correlation between antioxidant capacity and UA levels has also been observed in PD patients. Additionally, previous studies have reported strong associations between UA and serum albumin levels, and hypoalbuminemia is a well-established predictor of mortality in dialysis patients.[8,17-19] This paradoxical relationship may also be explained, at least in part, by the phenomenon of reverse epidemiology commonly observed in dialysis populations. Unlike the general population, where high levels of UA and cholesterol are typically associated with adverse outcomes, in dialysis patients, higher levels of these parameters may reflect better nutritional reserves and are often linked to improved survival. Therefore, rather than being a direct cause of mortality, hypouricemia may reflect an adverse nutritional or clinical status in dialysis populations.[8,12,17-19] The data suggest that low UA levels were associated with increased mortality in PD patients; however, time-averaged CRP did not differ significantly between survivors and non-survivors and was not associated with mortality in Cox regression analysis. In addition, the correlation analysis did not demonstrate a significant direct correlation between time-averaged UA and time-averaged CRP. Consequently, low UA should not be interpreted as a direct inflammatory marker based on the results of the present study. Instead, it may reflect a broader adverse clinical or nutritional profile in PD patients. Further studies are needed to clarify the underlying mechanisms.

There may be multiple reasons for the discrepant findings between UA levels and mortality. Uric acid levels are known to change significantly in the early stages of chronic kidney disease, particularly during the predialysis phase. Once dialysis is initiated, both HD and PD effectively clear UA, but UA levels can fluctuate over time and according to dialysis-related factors.[20,21] The timing of UA measurement is crucial, and relying on a single time-point measurement can be misleading. For example, UA levels measured shortly after PD initiation may still reflect the predialysis phase. In contrast, time-averaged UA values, assessed over longer periods, can account for these fluctuations.[9,12,22-25] This study adds to this body of evidence by demonstrating that low time-averaged UA levels are associated with increased mortality risk, highlighting the value of longitudinal monitoring over single-point measurements. Studies on the relationship between UA levels and mortality in PD patients are fewer than in HD patients, with inconsistent findings.[9,24,25] These differences may be attributed to variations in patient populations. The first study to assess the mortality risk associated with time-averaged UA values in PD patients, by Chang et al,[12] found that both high UA levels (≥8 mg/dL) and low UA levels (<6 mg/dL) were linked to increased mortality risk. Consistent with these findings, a recent study in PD patients reported that the lowest mortality risk was observed at time-averaged serum UA levels between approximately 5.65 and 7.13 mg/dL, suggesting a potential optimal range associated with improved survival.[26] The present findings similarly suggest that low time-averaged UA levels are associated with a higher risk of death, with survival analyses identifying a critical threshold of 5.75 mg/dL, below which mortality risk appeared to be higher. Establishing such a threshold may be of potential clinical relevance, as it could aid in identifying high-risk patients and informing risk

Table 3. Exploratory Sensitivity Analysis Around the Receiver Operating Characteristic Curve–Derived Uric Acid Cutoff

UA Cutoff	Low-UA Group, n	Deaths in Low-UA Group, n/N	High-UA Group, n	Deaths in High-UA Group, n/N	Log-Rank, *P*	Univariate Cox HR	95% CI	*P*
5.75 mg/dL (ROC-derived primary cutoff)	24	7	26	3	.009	**0.19**	**0.04-0.75**	**.01**
5.5 mg/dL (exploratory sensitivity cutoff)	15	4	35	6	.208	0.54	0.12-1.61	.22
6.0 mg/dL (exploratory sensitivity cutoff)	25	8	25	2	.041	0.246	0.05-1.196	.08

The 5.75 mg/dL threshold represents the primary ROC-derived cutoff. The 5.5 and 6.0 mg/dL thresholds were evaluated as exploratory adjacent sensitivity cutoffs.Bold values indicate statistical significance ($P < 0.05$).
HR, hazard ratio; UA, uric acid.

stratification; however, these findings should be interpreted with caution and confirmed in future studies.

In addition, exploratory sensitivity analyses using adjacent rounded thresholds of 5.5 and 6.0 mg/dL demonstrated a similar direction of association, with lower UA levels showing numerically higher mortality across these thresholds**.** Although statistical significance was attenuated at alternative cutoff values, likely because of the limited sample size and small number of mortality events, these findings support the exploratory stability of the ROC-derived threshold. These observations provide an additional perspective on the prognostic role of UA in PD, complementing previous inconsistent findings. While the current results highlight the potential clinical relevance of low UA levels, further large-scale, prospective studies are essential to confirm and extend these associations.

There are several important limitations to consider in this study. First, the small sample size of only 50 patients, with 10 mortality events, limits the statistical power of the analyses and reduces the precision and robustness of the findings and may increase the risk of overfitting in multivariable analyses. Second, the single-center and retrospective design introduces the risk of selection bias and unmeasured confounding, thereby limiting the generalizability of the results. Although biochemical markers such as serum albumin were available, a comprehensive and structured assessment of nutritional status—such as objective measures of muscle mass, sarcopenia, or frailty—was not performed, which may have contributed to residual confounding and potential overestimation of effect sizes. Third, cause-specific mortality was not analyzed, preventing the authors from determining

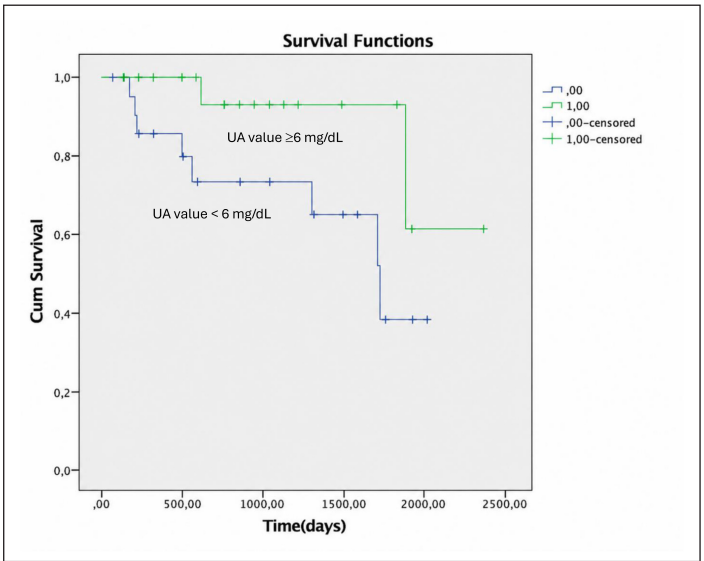

Figure 3 . Kaplan–Meier survival curve according to the 6.0 mg/dL UA cutoff. Patients with time-averaged UA levels <6.0 mg/dL showed poorer survival compared with those with UA levels ≥6.0 mg/dL (log-rank $P = .041$). For the 6.0 mg/dL cutoff, sensitivity and specificity for mortality prediction were 80% and 38%, respectively. UA, uric acid.

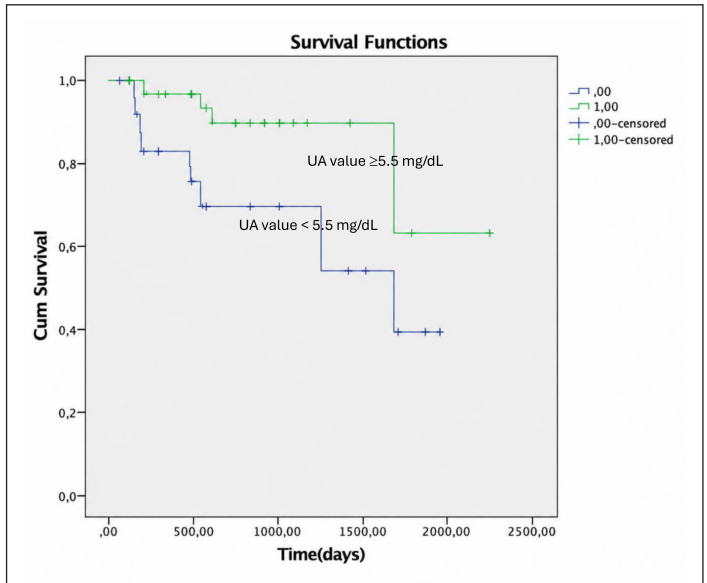

Figure 4. Kaplan–Meier survival curve according to the 5.5 mg/dL UA cutoff. Patients with time-averaged UA levels <5.5 mg/dL showed numerically poorer survival compared with those with UA levels ≥ 5.5 mg/dL; however, the difference did not reach statistical significance (log-rank $P = .208$). The 5.5 mg/dL threshold had a sensitivity of 60% and specificity of 25% for mortality prediction. UA, uric acid.

whether low UA is more strongly associated with cardiovascular, infectious, or malnutrition-related deaths. Additionally, the relatively short follow-up period may not fully capture long-term outcomes. Fourth, although time-averaged urine output was evaluated, a formal longitudinal trajectory analysis of residual urine output could not be performed because of the retrospective design and limited sample size. Fifth, despite the exclusion of patients receiving allopurinol, losartan, or furosemide, the potential influence of other medications affecting serum UA levels could not be completely eliminated. These limitations highlight the need for larger, prospective, multicenter studies with comprehensive adjustment for confounders to clarify the prognostic role of UA in PD patients, and indicate that the present findings should be interpreted cautiously.

In conclusion, this study suggests that persistently reduced time-averaged UA levels during PD follow-up are associated with a higher mortality risk. Rather than acting as a direct pathogenic factor, low UA may reflect a broader adverse clinical and nutritional profile in PD patients. These observations should be viewed as exploratory, given the limited sample size and retrospective nature of the study. Ongoing debate in the literature underscores the need for well-designed multicenter prospective studies to clarify the prognostic significance and biological interpretation of UA in this setting. Until such evidence is available, serial assessment of UA levels may provide clinicians with an additional supportive clue for identifying vulnerable patients.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Artificial Intelligence Usage Statement: The authors declared that no Artificial Intelligence Tool was used in the preparation of the manuscript.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of Ankara Dışkapı Yıldırım Beyazıt Education and Research Hospital (Approval No.: 149/01; Date: August 29, 2022).

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