DOI: 10.14744/cm.2025.85547
Comprehensive Medicine 2025;17(3):195-200

RESEARCH ARTICLE

Serum Uric Acid as a Biomarker for Survival in Solid Cancer Patients

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ABSTRACT

Objective: Serum uric acid (SUA), the end product of purine metabolism, has been implicated in cancer as both an antioxidant and pro-oxidant. Emerging evidence suggests that SUA may serve as a prognostic biomarker, particularly for overall survival (OS). This study evaluated the prognostic significance of SUA in patients with solid tumors.

Materials and Methods: A retrospective analysis was conducted on 132 patients with solid malignancies (breast, colon, rectum, pancreas, and gastric) treated at a single center between August 2023 and December 2024. SUA levels were measured pre- and post-chemotherapy. Patients were categorized into high vs. low SUA groups based on median values (4.3 mg/dL pre-treatment, 4.2 mg/dL post-treatment). OS was estimated using the Kaplan–Meier method and compared via log-rank tests.

Results: At a median follow-up of 21 months, 25.0% of patients had died. Baseline SUA above 4.3 mg/dL was associated with significantly shorter median OS (9.6 months [95% CI 7.6–11.6]) compared to SUA ≤4.3 mg/dL (12.1 months [10.1–14.3]; p=0.03). A similar survival detriment was observed for high post-chemotherapy SUA (>4.2 mg/dL: 9.6 months vs. ≤4.2:12.1 months, p=0.03). Traditional prognostic factors such as stage, liver metastasis, and sex were not statistically significant.

Conclusion: Elevated SUA is associated with worse OS in solid tumors and may serve as a practical, low-cost prognostic biomarker. Its consistent association across tumor types supports its integration into risk models. Prospective studies are needed to validate these findings and clarify underlying mechanisms.

Keywords: Cancer survival, solid tumors, uric acid

How to cite this article: Erinç O, Göktaş Aydın S, Yılmaz Ö, Aydın A, Aydın FB, Aydın Yoldemir Ş. Serum Uric Acid as a Biomarker for Survival in Solid Cancer Patients. CM 2025;17(3):195-200

INTRODUCTION

Serum uric acid (SUA), the final product of purine metabolism, is primarily synthesized in the liver and intestinal mucosa and is predominantly excreted by the kidneys.[1] Although traditionally linked to gout and kidney disease, SUA has more recently been implicated in cancer biology due to its paradoxical role in redox regulation.[2] As an antioxidant, SUA can neutralize reactive oxygen and nitrogen species, potentially limiting oxidative DNA damage and lipid peroxidation—both central mechanisms in carcinogenesis.[3] However, SUA may also exert pro-oxidant effects, especially when intracellular levels rise or in the presence of transition metals, contributing to oxidative stress, inflammation, and endothelial dysfunction.[4–6] These dual effects raise questions about the context-dependent influence of SUA on cancer development and outcomes.[7–9]

Recent studies have suggested a possible link between SUA levels and both cancer incidence and prognosis.[10] In colorectal cancer, high pre-treatment SUA levels were significantly associated with shorter progression-free and overall survival (OS) in patients receiving chemotherapy.[11] Similar observations were reported in breast cancer, where elevated SUA correlated with higher mortality and reduced treatment response.[12] Metastatic breast cancer patients'

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Received date: 01.07.2025
Revised date: 23.07.2025
Accepted date: 24.07.2025
Online date: 05.08.2025

survival has also been linked to uric acid levels: in a 2022 analysis conducted by Yilmaz et al.,[13] SUA emerged as an independent predictor of shorter progression-free and OS in patients receiving CDK4/6 inhibitors. Rao et al.[14] demonstrated that pre-immunotherapy SUA levels exceeding approximately 4.36 mg/dL were independently associated with a more than threefold increase in the hazard of death among patients with primary liver cancer undergoing immunotherapy (HR: 3.131; 95% CI: 1.766–5.553; p<0.001). Conversely, a U-shaped relationship has been proposed in prostate cancer, where both hypo- and hyperuricemia are associated with worse survival among patients on androgen deprivation therapy.[15]

The potential biological mechanisms behind these associations are multifaceted. Elevated SUA may reflect increased tumor burden or cell turnover, leading to higher rates of nucleic acid degradation and purine metabolism.[16] Additionally, SUA may activate the NOD-like receptor family pyrin domain-containing 3 (NLRP3) inflammasome, thereby enhancing proinflammatory cytokine production (e.g., IL-1β, IL-6), which has been linked to cancer proliferation and metastasis.[17,18] These findings suggest that the prognostic significance of SUA may vary by cancer type, stage, treatment modality, and comorbid conditions.

To address this knowledge gap, the present study evaluates the prognostic significance of SUA levels measured at both baseline and after systemic chemotherapy in a diverse cohort of patients with solid malignancies, including breast, colorectal, gastric, and pancreatic cancers. Unlike prior studies limited to single tumor types or pre-treatment SUA values,[11–14] we examine dynamic changes in SUA and their association with OS. We hypothesize that elevated baseline or persistently high post-treatment SUA levels would be independently associated with adverse survival outcomes. By clarifying this relationship, our findings may support the integration of SUA into risk stratification frameworks and highlight its potential role as a clinically accessible biomarker that reflects tumor burden, treatment response, or therapy-related metabolic stress.

MATERIALS and METHODS

Study Design and Settings

This retrospective cohort study included patients who presented to our center's medical oncology outpatient clinic between August 2023 and December 2024. The medical records of all patients were reviewed from the institutional database. Demographic and clinical variables, including age, sex, cancer type, stage at diagnosis, presence of liver metastases, and comorbid diabetes mellitus, were recorded for all patients. Patients with incomplete clinical or laboratory data were excluded from the study. One hundred thirty-two patients with complete data sets were included in the final analysis.

The exclusion criteria were as follows: active gout or hyperuricemia requiring treatment, chronic kidney disease stage ≥ 3 (eGFR < 60 ml/min/1.73 m^2), concomitant use of uric acid–modulating medications, high-dose salicylates, and loop or thiazide diuretics.

Ethics Approval and Consent to Participate

This study was carried out in compliance with the Declaration of Helsinki. Informed consent was obtained from all patients or their caregivers. The Local Ethics Committee of Istanbul Medipol University approved the study (decision date: 20.06.2025, number: E-10840098-202.3.02-3811).

Serum Uric Acid Measurement

SUA was measured at two time points: before the initiation of systemic chemotherapy (pre-treatment) and after completion of first-line therapy or during follow-up (post-treatment). All SUA measurements were performed in our hospital's clinical laboratory using the Roche/cobas 8000 c 702 analyzer with the Roche Elecsys Uric Acid assay kit (Roche Diagnostics, Mannheim, Germany). We dichotomized patients into "low" vs. "high" SUA groups based on the median SUA values of our cohort: the median pre-treatment SUA was 4.3 mg/dL, and the median post-treatment SUA was 4.2 mg/dL. These cutoffs were used to define low (≤ median) and high (> median) SUA subgroups for survival comparisons.

Outcomes

The primary outcome of this study was to evaluate the association between SUA levels, both pre-treatment and post-treatment, and OS in patients with solid tumors.

As part of our secondary outcomes, we compared OS across different cancer types to assess whether tumor origin influenced prognosis in the context of SUA levels. Additionally, we examined whether clinical variables such as sex, the presence of diabetes mellitus, and hepatic metastasis were associated with OS.

Statistical Analysis

Statistical analyses were performed using SPSS version 24 (IBM Corp., Armonk, NY). The distribution of continuous variables was assessed using the Kolmogorov–Smirn-

Table 1. Demographic and clinical characteristics of the patients

Patient characteristic (n=132)	Value	
	n	%
Age, years	61 (24–86)	
Gender		
Female	67	50.8
Male	65	49.2
Cancer type		
Pancreatic	18	13.6
Rectal	11	8.3
Colon	41	31.1
Gastric	24	18.2
Breast	38	28.8
Cancer stage		
Stage 1	6	4.5
Stage 2	32	24.2
Stage 3	53	40.2
Stage 4	41	31.1
Presence of hepatic metastasis	26	19.7
Exitus	33	25.0

ov test, and none were normally distributed. Therefore, non-parametric tests were employed for comparisons. SUA values were categorized based on median cut-off levels determined from the dataset (4.3 mg/dL for pre-treatment and 4.2 mg/dL for post-treatment). OS was defined as the time from initial cancer diagnosis to death or the last known follow-up. Survival analyses were conducted using Kaplan–Meier estimates, and differences in survival between groups were evaluated using the log-rank test. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 132 cancer patients were included, with key demographics and clinical features summarized in Table 1. The median age was 61 years (IQR 53–68). There were 67 females (50.8%) and 65 males (49.2%). By cancer type, the cohort comprised 41 colon cancer (31.1%), 38 breast cancer (28.8%), 24 gastric cancer (18.2%), 18 pancreatic cancer (13.6%), and 11 rectal cancer (8.3%) patients. Disease stage at diagnosis varied: 6 patients (4.5%) had stage I, 32 (24.2%) stage II, 53 (40.2%) stage III, and 41 (31.1%) stage IV disease. A subset of 26 patients (19.7%) had documented hepatic metastases at presentation. Comorbid diabetes mellitus was present in 21 patients (15.9%). At the time of analysis, 33 patients (25.0%) had died, whereas 99 were alive or lost to follow-up. The median follow-up duration was 21 months.

We observed a significant inverse relationship between SUA levels and OS. Patients with low pre-treatment SUA (≤4.3 mg/dL) had a longer median OS compared to those with high pre-treatment SUA (>4.3 mg/dL). At a median follow-up of 21 months, the median OS in the low baseline SUA group was 12.1 months (95% confidence interval [CI] 10.1–14.3), whereas the high SUA group had a median OS of 9.6 months (95% CI 7.6–11.6). This difference was statistically significant (p=0.036 by log-rank test). Pre- and post-chemotherapy SUA levels did not change significantly (p=0.6). Figure 1 illustrates the Kaplan–Meier survival curves stratified by pre-chemotherapy SUA, demonstrating the separation of outcomes between the two groups.

We found a similar prognostic pattern for post-treatment SUA levels. Patients whose SUA remained low after chemotherapy (≤4.2 mg/dL) had better survival than those with post-treatment hyperuricemia (>4.2 mg/dL). Median OS was 12.1 months in the low post-treatment SUA group vs. 9.6 months in the high post-SUA group (log-rank p=0.03). Figure 2 shows the post-treatment SUA survival curves, which closely paralleled the pre-treatment SUA findings. Notably, the absolute difference in median OS between low and high SUA groups was approximately 2.5–2.6 months for both time points.

When comparing OS across cancer types, adjusted for SUA levels (cut-off: 4.3 mg/dL), significant differences were observed. Specifically, rectal cancer patients exhibited significantly shorter survival than those with colon cancer (χ^2=5.338, p=0.021), gastric cancer (χ^2=6.040, p=0.014), and breast cancer (χ^2=8.578, p=0.003). However, the survival differences between rectal and pancreatic cancers (p=0.072) and between pancreatic and other tumor types were not statistically significant (p>0.05 for all comparisons).

Cancer type was a significant determinant of OS (p=0.01), reflecting the heterogeneous prognoses of different malignancies. Gastric cancer patients had the longest survival in our dataset, with a median OS of approximately 12.7 months (95% CI 6.4–18.9), followed closely by breast cancer patients at 12.2 months (95% CI 8.6–15.6). In contrast, patients with rectal cancer had the poorest outcomes, with a median OS of only 5.3 months (95% CI 2.1–8.5). The median OS for colon cancer patients was 9.6 months (95% CI 6.6–12.5), and for pancreatic cancer, 11.9 months (95% CI 8.5–12.2).

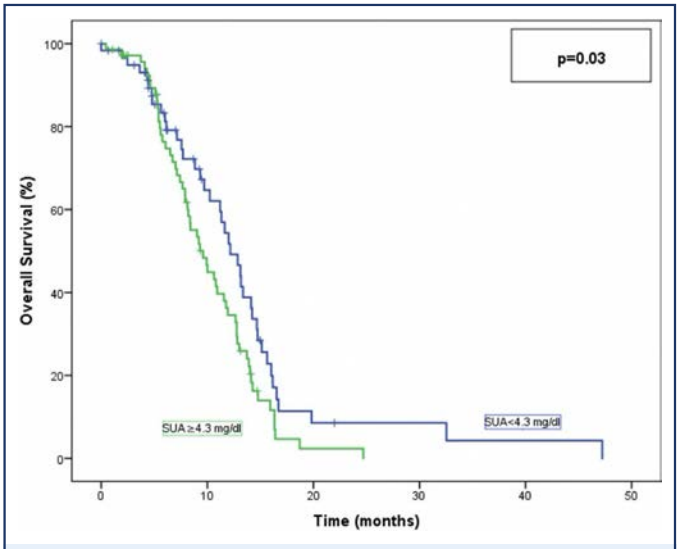

Figure 1. Kaplan–Meier overall survival curves for patients stratified by baseline (pre-chemotherapy) serum uric acid levels (cut-off=4.3 mg/dL). The blue curve represents patients with SUA <4.3 mg/dL, and the green curve represents those with SUA ≥4.3 mg/dL

SUA: Serum uric acid

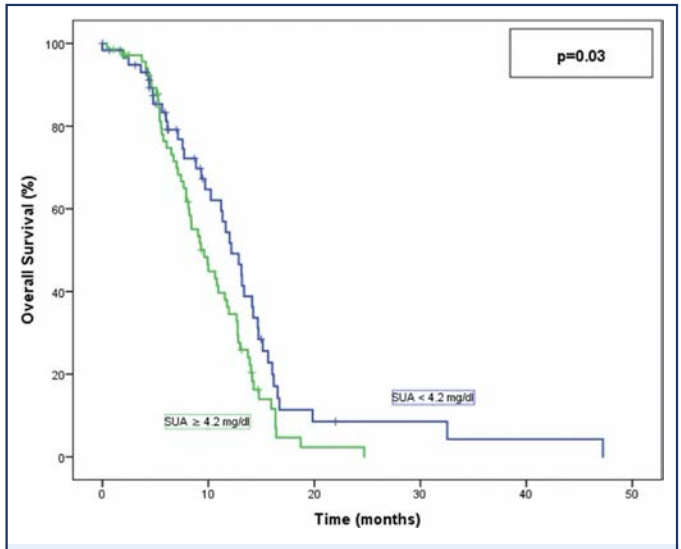

Figure 2. Kaplan–Meier overall survival curves based on post-chemotherapy serum uric acid levels (cut-off=4.2 mg/dL). The blue line indicates patients with post-treatment SUA <4.2 mg/dL, and the green line those with SUA ≥4.2 mg/dL

Disease stage at diagnosis showed a non-significant trend toward worse survival in stage IV patients compared to those with stage I–III (p=0.10).

The presence of liver metastases at diagnosis was associated with shorter OS—median OS was 8.4 months in patients with liver metastases (n=26, 19.7%) versus 11.5 months in those without (n=67, 50.8%)— though this difference did not reach statistical significance (p=0.08). Neither patient sex (female: n=67, 50.8%; male: n=65, 49.2%; p=0.4) nor diabetes mellitus status (n=21, 15.9%; p=0.5) showed a significant impact on survival in this cohort.

DISCUSSION

This study investigated the prognostic relevance of serum SUA levels measured before and after systemic chemotherapy in patients with solid tumors, including breast, gastric, pancreas, colon, and rectum. Our findings demonstrate that both elevated baseline and post-treatment SUA levels are associated with significantly reduced OS. Specifically, patients with pre-treatment SUA above 4.3 mg/dL had a median OS of 9.6 months compared to 12.1 months in those with lower levels. A similar trend was observed post-treatment, where SUA above 4.2 mg/dL corresponded to significantly shorter survival.

Our findings align with several prior studies that reported similar associations between hyperuricemia and adverse cancer outcomes. Yue et al.[19] demonstrated that in a cohort of 443 breast cancer patients, high SUA concentrations were significantly associated with reduced OS (HR 2.13, 95% CI 1.15–3.94, p=0.016), and SUA emerged as an independent prognostic factor in multivariate analysis. Similarly, Chen et al.[20] found that the preoperative level of SUA was a strong predictor of OS and disease-free survival (DFS) in patients undergoing esophagectomy for esophageal squamous cell carcinoma. Patients with a pre-operative SUA level >304.5 μmol/L had significantly shorter DFS and OS than patients with a pre-operative SUA level ≤304.5 μmol/L (for DFS 58 vs. 99 months, p<0.001, and for OS 64 vs. 104 months, p<0.001, respectively). In advanced hepatocellular carcinoma, Wu et al.[21] reported that patients with elevated SUA levels (> 360 μmol/L) had significantly shorter median survival (133.5 vs. 176.0 days, p=0.0013), and this was associated with increased oxidative stress and xanthine oxidase activity in tumor tissues. In a study by Yan et al.,[22] hyperuricemia was significantly associated with poorer OS and DFS in colon cancer patients (log-rank p=0.0008). It was an independent prognostic factor for OS in both univariate (HR=2.09, p=0.002) and multivariate analyses (HR=1.94, p=0.005). Zhou et al.[23]'s retrospective study found survival outcomes varied significantly across SUA tertiles (p<0.05), with higher SUA levels associated with worse prognoses in patients with hepatoblastoma. The 5-year event-free survival rates were 90.3%, 83.3%, and 66.2%, while the 5-year OS rates were 97.0%, 88.7%, and 80.0% from the lowest to highest SUA tertile, respectively. These findings re-

inforce the view that SUA may serve as a robust and broadly applicable prognostic biomarker across diverse malignancies.

Our subgroup analysis revealed significant survival differences across cancer types after adjustment for serum SUA levels, with rectal cancer patients exhibiting markedly shorter OS compared to those with colon, gastric, or breast cancers. This disparity may reflect distinct tumor biological behaviors and the metabolic role of uric acid among different tumor types. Saidak et al.[24] conducted a pan-cancer transcriptomic study demonstrating heterogeneous expression of uricogenesis-related genes across tumors, including key enzymes and transporters like XDH, ABCG2, and SLC2A9, which regulate intracellular uric acid levels and redox state. Their findings suggest that tumor-intrinsic regulation of uric acid may affect prognosis independently of systemic SUA, helping to explain why the prognostic impact of SUA is not uniform across malignancies. Although pancreatic cancer is typically associated with the poorest prognosis, we found no significant difference between pancreatic and rectal cancers, likely reflecting the similarly aggressive nature of both tumor types and the lack of substantial therapeutic advancements for pancreatic adenocarcinoma.

Despite the well-known association between cancer stage and survival,[25,26] this study couldn't demonstrate a significant difference between stages (p=0.10). This discrepancy may be attributed to tumor-type heterogeneity within our cohort and the limited sample size in each staging subgroup, which may have reduced the statistical power to detect stage-specific survival differences.

Several limitations should be acknowledged. First, the retrospective, single-center design may introduce selection bias and limit generalizability. Randomized controlled trials or Mendelian randomization analyses could help determine whether uric acid plays a causal and modifiable role in cancer progression or simply reflects underlying disease severity. The small sample size in each group affected the statistical power. Furthermore, due to the retrospective nature of the study and lack of standardized follow-up for recurrence or progression, we were unable to evaluate DFS, which is an important oncologic endpoint.

On the other hand, a key contribution of this study lies in its evaluation of dynamic changes in SUA levels before and after systemic chemotherapy, along with their respective associations with OS. Unlike prior research focused solely on baseline SUA, we demonstrated that SUA levels remain largely stable throughout chemotherapy, suggesting limited treatment-related modulation, which suggests SUA may be a time-independent prognostic biomarker in solid tumor patients.

CONCLUSION

This study demonstrates that elevated SUA levels, both before and after chemotherapy, are significantly associated with reduced survival in patients with solid tumors. As a cost-effective and widely available laboratory test, SUA may serve as a practical adjunct for risk stratification in cancer care. These results underscore the need for future, larger, prospective multicenter cohort studies and mechanistic investigations to elucidate the biological role of uric acid in cancer progression.

Disclosures

Ethics Committee Approval: The study was approved by the Istanbul Medipol University Non-interventional Clinical Research Ethics Committee (No: E-10840098-202.3.02-3811, Date: 20/06/2025).

Informed Consent: Informed consent was obtained from all participants.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study received no financial support.

Use of AI for Writing Assistance: No AI technologies utilized.

Author Contributions: Concept – O.E, S.G.A.; Design – O.E., S.G.A.; Supervision – Ş.A.Y.; Funding – O.E.; Materials – S.G.A., Ö.Y., F.B.A., A.A.; Data collection and/or processing – O.E., S.G.A., Ö.Y., F.B.A., A.A., Ş.A.Y.; Data analysis and/or interpretation – O.E., A.A., S.G.A.; Literature search – O.E., S.G.A., Ö.Y.; Writing – O.E.; Critical review – O.E.

Peer-review: Externally peer-reviewed.

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