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Baseline Serum Albumin-to-Creatinine Ratio as an Independent Prognostic Marker in *De Novo* Metastatic HER2-negative Gastric Cancer

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ABSTRACT

Objective: Systemic inflammation, nutritional status, and metabolic reserve are recognized determinants of survival in metastatic gastric cancer patients. The serum albumin-to-creatinine ratio (ACR) combines nutritional and renal/metabolic information, but its prognostic value in gastric cancer remains unclear. We aimed to investigate the association between baseline ACR and survival outcomes in patients with *de novo* metastatic human epidermal growth factor receptor 2 (HER2)-negative gastric cancer.

Material and Methods: This retrospective, single-center study included 112 adults with histologically confirmed *de novo* metastatic HER2-negative gastric cancer diagnosed between April 2011 and January 2024. Baseline serum albumin and creatinine levels obtained within 15 days prior to treatment initiation were used to calculate the ACR. Patients were categorized into low and high ACR groups according to the cohort median. Progression-free survival (PFS) and overall survival (OS) were analyzed using the Kaplan-Meier method and Cox proportional hazards models.

Results: The median follow-up duration was 40.6 months. The median PFS for the entire cohort was 6.8 months, and the median OS was 13.7 months. Patients with high ACR had significantly longer PFS (7.2 vs. 6.0 months; p=0.005) and OS (16.9 vs. 10.2 months; p<0.001) than those with low ACR. In multivariable analysis, high ACR remained independently associated with improved PFS [hazard ratio (HR): 0.65; 95% confidence interval (CI): 0.44-0.97; p=0.034) and OS (HR: 0.38; 95% CI: 0.21-0.67; p<0.001)]. Eastern Cooperative Oncology Group performance status ≥1 was also independently associated with worse survival outcomes.

Conclusion: Baseline serum ACR is an independent prognostic marker for both PFS and OS in patients with *de novo* metastatic HER2-negative gastric cancer. As a simple and routinely available laboratory parameter, ACR may contribute to risk stratification in advanced disease.

Keywords: Gastric cancer; metastatic disease; albumin-to-creatinine ratio; prognosis; survival

INTRODUCTION

Gastric cancer (GC) is one of the leading causes of cancer-related mortality worldwide, with a substantial proportion of patients presenting with advanced or metastatic disease at the time of diagnosis.[1] In *de novo* metastatic GC, curative treatment is not feasible, and systemic therapy is the main therapeutic approach. Although the incorporation of combination chemotherapy regimens and, more recently, targeted agents and immune checkpoint inhibitors has modestly improved survival outcomes, the prognosis for metastatic disease remains poor and varies considerably among patients.[2-5] This clinical variability underscores the need for readily available, reproducible prognostic biomarkers that may facilitate risk stratification, guide treatment decisions, and support individualized decision-making in clinical practice.

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Cancer-associated systemic inflammation and nutritional impairment are increasingly recognized as key determinants of tumor progression and survival across malignancies.[6,7] In GC, several inflammation- and nutrition-based biomarkers derived from routine laboratory parameters, such as C-reactive protein (CRP), serum albumin, neutrophil-to-lymphocyte ratio, modified Glasgow prognostic score, and CRP-to-albumin ratio (CAR), have been associated with overall survival (OS).[8-10] Serum albumin reflects both nutritional status and systemic inflammatory activity, and reduced levels have consistently been associated with poor oncologic outcomes.[11,12] Renal function parameters, including serum creatinine and estimated glomerular filtration rate (eGFR), have also been associated with unfavorable outcomes in various malignancies; however, data specifically addressing their prognostic role in metastatic GC remain limited.[13,14] These observations suggest that biomarkers integrating inflammatory, nutritional, and metabolic dimensions may provide complementary prognostic information for advanced disease.

The albumin-to-creatinine ratio (ACR) integrates two routinely measured laboratory parameters that reflect complementary aspects of the host condition, including nutritional status and renal function. By combining these variables into a single index, the ACR may provide a concise and clinically practical summary of systemic status. In recent years, low ACR levels have been associated with inferior survival across common solid tumors and have also been reported as a prognostic indicator in specific clinical settings, such as liposarcoma and patients treated with immune checkpoint inhibitors.[15-17] However, the prognostic significance of ACR has not yet been examined in GC. Therefore, we aimed to investigate the association between baseline serum ACR and survival outcomes in patients with *de novo* metastatic human epidermal growth factor receptor 2 (HER2)-negative GC and to assess its potential utility as an accessible prognostic biomarker in this population.

MATERIAL AND METHODS

Patient Population and Data Collection

This retrospective, single-center study included adult patients (≥18 years) with histologically confirmed, *de novo* metastatic HER2-negative GC diagnosed between April 2011 and January 2024. All eligible patients received first-line platinum- and fluoropyrimidine-based systemic therapy.

As part of the inclusion criteria, patients were required to have baseline laboratory measurements, including serum albumin and creatinine levels, obtained within 15 days prior to the initiation of systemic therapy. Patients were excluded if baseline laboratory data required for ACR calculation were unavailable, if clinical or radiological follow-up data were insufficient, or if a synchronous malignancy other than GC was present. Patients with an eGFR <60 mL/min/1.73 m^2 at treatment initiation were excluded to minimize the potential confounding effects of impaired renal function on ACR measurements. In addition, patients meeting the Kidney Disease: Improving Global Outcomes criteria for acute kidney injury at treatment initiation were excluded.[18] eGFR was estimated using the Chronic Kidney Disease Epidemiology Collaboration equation.[19]

Demographic and clinical variables, including age, sex, Eastern Cooperative Oncology Group (ECOG) performance status, tumor location, and metastatic sites, were obtained from medical records. Baseline laboratory parameters included serum albumin, creatinine, lactate dehydrogenase (LDH), carcinoembryonic antigen (CEA), and hemoglobin levels. The ACR was calculated as the ratio of serum albumin (g/dL) to serum creatinine (mg/dL). As no established cut-off value has been defined for ACR, patients were categorized into low- and high-ACR groups based on the cohort median.

Definition of Survival Outcomes

Progression-free survival (PFS) was defined as the time from the initiation of first-line systemic therapy to radiologically confirmed disease progression or death from any cause, whichever occurred first. Patients without documented disease progression or death were censored on the date of their last follow-up visit.

OS was defined as the time from the initiation of first-line systemic therapy to death from any cause. Patients who were alive at the last follow-up were censored on the date of their last visit.

Ethical Considerations

This study was conducted in accordance with the Declaration of Helsinki and approved by the İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine Ethics Committee (approval number: 1064826; date: 14.08.2024). Given the retrospective nature of this study, the requirement for informed consent was waived.

Statistical Analysis

All statistical analyses were conducted using the IBM SPSS Statistics software, version 27.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed variables were expressed as mean ± standard deviation, whereas non-normally distributed variables were presented as median with interquartile range (IQR). Comparisons between groups

were performed using the independent samples t-test or the Mann-Whitney U test, as appropriate. Categorical variables were summarized as frequencies and percentages and were compared using the chi-square test or Fisher's exact test when necessary.

PFS and OS were estimated using the Kaplan-Meier method, and differences between groups were evaluated using the log-rank test. Univariate Cox proportional hazards regression analyses were performed to assess the association between baseline clinical and laboratory variables and the survival outcomes. Variables with a p-value <0.10 in the univariate analyses were included in the multivariate Cox regression models. Hazard ratios (HRs) and corresponding 95% confidence intervals (CIs) were calculated for each outcome. A two-sided p-value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics

A total of 112 patients with *de novo* metastatic HER2-negative GC were included in the analysis. The median baseline ACR was 49.6 (IQR: 41.3-58.4). Patients were categorized into low- and high-ACR groups based on the cohort median (n=56 in each group).

The median age of the entire cohort was 58 years (IQR: 51-67.8 years). The age distribution was comparable between the low and high ACR groups; however, the sex distribution differed significantly, with a higher proportion of female patients in the high ACR group (p<0.001).

All other baseline demographic, clinical, and laboratory variables, including ECOG performance status, body mass index, tumor-related characteristics, metastatic burden, and key laboratory parameters, were similarly distributed between the two groups, suggesting a largely homogeneous baseline profile. The detailed demographic, clinical, and laboratory characteristics of the study population are summarized in Table 1.

All patients received fluoropyrimidine- and platinum-based first-line chemotherapy. The distribution of treatment regimens according to the ACR groups is shown in Table 2.

Survival Outcomes According to ACR

The median follow-up duration for the entire cohort was 40.6 months (95% CI: 30.6-50.7). During the follow-up period, 108 (96.4%) patients experienced disease progression, and 102 (91.1%) died.

The median PFS of the entire cohort was 6.8 months (95% CI: 6.0-7.5). When participants were stratified by ACR group, the median PFS was 6.0 months (95% CI: 4.8-7.3) in the low ACR group and 7.2 months (95% CI: 5.7-8.7) in the high ACR group. The difference between the two groups was statistically significant (p=0.005) (Figure 1).

In the univariate Cox regression analysis for PFS, ECOG PS (≥1 vs. 0) was associated with worse PFS (HR: 1.88; 95% CI: 1.19-2.97; p=0.007), whereas high ACR was associated with improved PFS (HR: 0.58; 95% CI: 0.39-0.86; p=0.006). Primary tumor location [stomach vs. gastroesophageal junction (GEJ)] showed a borderline association with worse PFS (p=0.064). In the multivariate model, ECOG PS (HR: 1.71; 95% CI: 1.07-2.75; p=0.026) and primary tumor location (HR: 1.64; 95% CI: 1.03-2.61; p=0.038) were independently associated with worse PFS, while high ACR remained independently associated with improved PFS (HR: 0.65; 95% CI: 0.44-0.97; p=0.034). The results of the univariate and multivariate Cox regression analyses are shown in Table 3.

The median OS for the entire cohort was 13.7 months (95% CI: 10.7-16.7). When stratified according to ACR groups, the median OS was 10.2 months (95% CI: 8.8-11.6) in the low ACR group and 16.9 months (95% CI: 15.6-18.3) in the high ACR group. The difference between the two groups was statistically significant (p<0.001) (Figure 2).

In the univariate Cox regression analyses, sex, ECOG PS, LDH status, and ACR were significantly associated with OS. Tumor location (stomach vs. GEJ) and CEA status showed a borderline association. In the multivariable model, ECOG PS ≥1 was independently associated with worse OS (HR: 2.14; 95% CI: 1.18-3.85; p=0.012), whereas high ACR remained independently associated with improved OS (HR: 0.38; 95% CI: 0.21-0.67; p<0.001). The other variables were not statistically significant. Detailed results of the univariate and multivariate Cox regression analyses for OS are presented in Table 4.

DISCUSSION

To our knowledge, this is the first study to evaluate the prognostic significance of the baseline serum ACR in patients with *de novo* metastatic HER2-negative GC. Our findings demonstrated that lower ACR levels were significantly associated with poorer PFS and OS, and that ACR remained an independent prognostic factor after adjustment for relevant clinical and laboratory variables. These results suggest that ACR, a composite marker reflecting baseline metabolic and nutritional status, may serve as a clinically meaningful prognostic indicator in advanced HER2-negative GC.

Albumin has a well-established role in the prognostic assessment of GC and constitutes a central component of several inflammation- and nutrition-based scores. Indices

TABLE 1: Baseline demographic, clinical, and laboratory characteristics according to ACR group.				
	All patients (n=112)	**Low ACR (n=56)**	**High ACR (n=56)**	**p-value**
Age (years), median (IQR)	58.0 (51-67.8)	60.5 (54.3-69.0)	55.5 (47.0-66.8)	0.065
Age category, n (%)				
<65 years	77 (68.8)	36 (64.3)	41 (73.2)	0.308
≥65 years	35 (31.3)	20 (35.7)	15 (26.8)	
Gender, n (%)				
Female	35 (31.3)	7 (12.5)	28 (50)	**<0.001**
Male	77 (68.8)	49 (87.5)	28 (50)	
ECOG PS, n (%)				
0	28 (25.0)	11 (19.6)	17 (30.4)	0.340
1	78 (69.6)	41 (73.2)	37 (66.1)	
2	6 (5.4)	4 (7.1)	2 (3.6)	
BMI (kg/m^2) median (IQR)	23.5 (20.8-26.1)	23.4 (21.7-26.9)	23.5 (20.0-26.0)	0.427
Primary tumor location, n (%)				
GEJ	28 (25.0)	13 (23.2)	15 (26.8)	0.663
Stomach	84 (75.0)	43 (76.8)	41 (73.2)	
Lauren classification, n (%)				
Diffuse	59 (52.7)	27 (48.2)	32 (57.1)	0.718
Intestinal	26 (23.2)	13 (23.2)	13 (23.2)	
Unknown	27 (24.1)	16 (28.6)	11 (19.6)	
Signet ring cell component, n (%)				
Absent	52 (46.4)	26 (46.4)	26 (46.4)	0.918
Present	49 (43.8)	24 (42.9)	25 (44.6)	
Unknown	11 (9.8)	6 (10.7)	5 (8.9)	
Number of metastatic sites, n (%)				
≤2 sites	72 (64.3)	36 (64.3)	36 (64.3)	1.000
>2 sites	40 (35.7)	20 (35.7)	20 (35.7)	
Metastatic sites, n (%)				
Liver metastasis	43 (38.4)	24 (42.9)	19 (33.9)	0.331
Lung metastasis	15 (13.4)	7 (12.5)	8 (14.3)	0.781
Peritoneal metastasis	47 (42.0)	25 (44.6)	22 (39.3)	0.566
CEA status				
≤ULN	58 (51.8)	27 (48.2)	31 (55.3)	0.648
>ULN	39 (34.8)	20 (35.7)	19 (33.9)	
Unknown	15 (13.4)	9 (16.1)	6 (10.7)	
LDH (U/L), median (IQR)	192 (154-272)	188.5 (152.5-257)	194 (154-305)	0.596
Hemoglobin (g/dL), mean ± SD	11.4±2.1	11.7±2.1	11.1±2.1	0.115
Albumin (g/dL), mean ± SD	3.90±0.50	3.86±0.48	3.93±0.52	0.464
Creatinine (mg/dL), mean ± SD	0.80±0.19	0.95±0.11	0.65±0.13	**<0.001**

ACR: Albumin-to-creatinine ratio; BMI: Body mass index; CEA: Carcinoembryonic antigen; ECOG: Eastern Cooperative Oncology Group; GEJ: Gastroesophageal junction; IQR: Interquartile range; LDH: Lactate dehydrogenase; SD: Standard deviation; ULN: Upper limit of normal.

TABLE 2: First-line treatment characteristics according to ACR group.

Treatment regimen	All patients (n=112)	Low ACR (n=56)	High ACR (n=56)
Oxaliplatin-based chemotherapy, n (%)	77 (68.8)	40 (71.4)	37 (66.1)
FOLFOX	67 (59.8)	35 (62.5)	32 (57.1)
CAPOX	8 (7.1)	4 (7.1)	4 (7.1)
FOLFOX + pembrolizumab	2 (1.8)	1 (1.8)	1 (1.8)
Cisplatin-based chemotherapy, n (%)	35 (31.2)	16 (28.6)	19 (33.9)
Cisplatin + 5-FU	24 (21.4)	13 (23.2)	11 (19.6)
DCF	5 (4.5)	1 (1.8)	4 (7.1)
Cisplatin + 5-FU + pembrolizumab	6 (5.4)	2 (3.6)	4 (7.1)

ACR: Albumin-to-creatinine ratio; FOLFOX: Fluorouracil, leucovorin, and oxaliplatin; CAPOX: Capecitabine and oxaliplatin; DCF: Docetaxel, cisplatin, and fluorouracil; 5-FU: 5-fluorouracil.

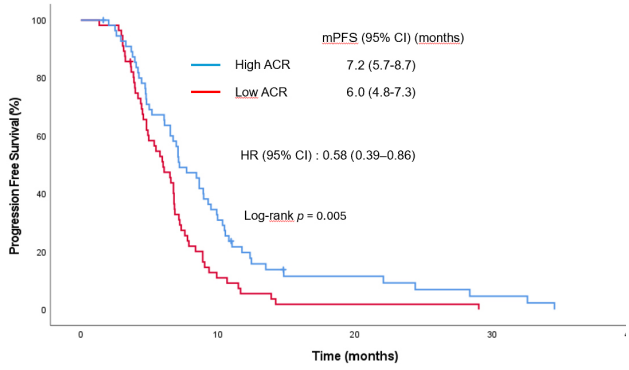

FIGURE 1: Kaplan-Meier curve for progression-free survival according to the ACR group in patients with *de novo* metastatic HER2-negative gastric cancer.

ACR: Albumin-to-creatinine ratio; CI: Confidence interval; HER2: Human epidermal growth factor receptor 2; PFS: Progression-free survival; HR: Hazard ratio

such as the CAR and the Prognostic Nutritional Index have been shown to correlate with survival outcomes in both early-stage and metastatic settings, underscoring the role of systemic inflammatory burden and nutritional reserve in disease progression.[10,11,20] However, these scores primarily focus on inflammatory and immune-related parameters.

In contrast, creatinine has received limited attention as a prognostic marker in metastatic GC, and data directly evaluating its association with survival in this setting remain scarce. Tanaka et al.[13] demonstrated in a surgically treated cohort of patients with locally advanced disease that preoperative eGFR was associated with postoperative complications and clinical outcomes. Although this population differs from *de novo* metastatic patients, the findings suggest that renal and metabolic reserves may influence oncologic outcomes.

In the metastatic setting, where cachexia and progressive muscle loss are common, parameters incorporating creatinine may capture an additional dimension of physiological

TABLE 3: Univariate and multivariate Cox regression analyses for progression-free survival.

	Univariate analyses		Multivariate analyses	
Variable	HR (95% CI)	p-value	HR (95% CI)	p-value
Age (per 1-year increase)	1.01 (0.99-1.02)	0.252		
Sex (male vs. female)	1.33 (0.87-2.02)	0.184		
ECOG PS (≥1 vs. 0)	1.88 (1.19-2.97)	0.007	1.71 (1.07-2.75)	0.026
BMI (per 1 kg/m^2 increase)	1.00 (0.95-1.04)	0.911		
Tumor location (stomach vs. GEJ)	1.55 (0.98-2.45)	0.064	1.64 (1.03-2.61)	0.038
Liver metastasis (present vs. absent)	1.20 (0.81-1.78)	0.365		
Lung metastasis (present vs. absent)	1.58 (0.91-2.75)	0.106	1.39 (0.79-2.45)	0.261
Peritoneal metastasis (present vs. absent)	0.79 (0.54-1.17)	0.244		
CEA status (>ULN vs. ≤ULN)	1.15 (0.76-1.75)	0.512		
LDH status (>ULN vs. ≤ULN)	1.35 (0.87-2.11)	0.187		
ACR group (high vs. low)	0.58 (0.39-0.86)	0.006	0.65 (0.44-0.97)	0.034

ACR: Albumin-to-creatinine ratio; BMI: Body mass index; CEA: Carcinoembryonic antigen; CI: Confidence interval; ECOG PS: Eastern Cooperative Oncology Group performance status; GEJ: Gastroesophageal junction; HR: Hazard ratio; LDH: Lactate dehydrogenase; ULN: Upper limit of normal.

vulnerability that is not reflected by inflammation-based markers.

Against this background, integrating albumin and creatinine into a composite index such as ACR may provide a broader assessment of host-related health status in advanced disease. The prognostic value of ACR has recently been explored in broader oncological settings. Zhao et al.[15] demonstrated that lower ACR levels were independently associated with inferior OS in a large cohort of patients with solid tumors, including gastrointestinal malignancies. In a disease-specific analysis, Panotopoulos et al.[16] reported that alterations in albumin and creatinine levels were associated with poor disease-specific survival in patients with liposarcoma. Similarly, Bas et al.[17] showed that a lower serum ACR independently predicted shorter progression-free and OS in patients treated with immune checkpoint inhibitors.

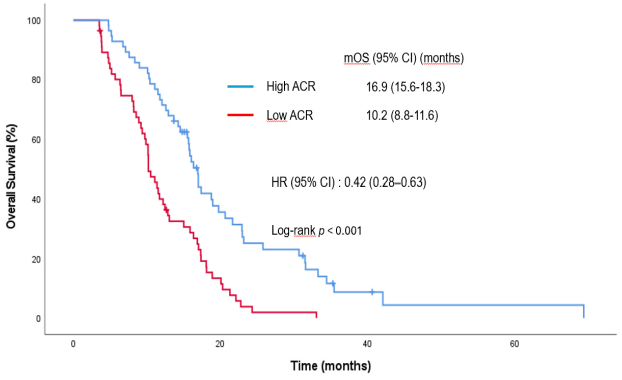

FIGURE 2: Kaplan-Meier curve for overall survival according to the ACR group in patients with *de novo* metastatic HER2-negative gastric cancer.

ACR: Albumin-to-creatinine ratio; CI: Confidence interval; HER2: Human epidermal growth factor receptor 2; OS: Overall survival; HR: Hazard ratio

Despite these findings, the ACR has not been previously examined in GC. By evaluating a homogeneous cohort of patients with *de novo* metastatic HER2-negative disease, our study adds disease-specific evidence to the existing literature. Notably, the association between ACR and OS appeared more pronounced than the association observed for PFS, which may suggest that ACR captures host-related determinants of long-term outcomes beyond early tumor response. This observation warrants further investigation in prospective studies.

In our cohort, the high ACR group included a higher proportion of female patients. However, sex did not retain independent prognostic significance in the multivariable analysis, suggesting that the survival differences associated with ACR were unlikely to be solely attributable to differences in sex distribution. Previous studies on metastatic GC have reported inconsistent findings regarding the prognostic impact of sex, and its role remains uncertain.[21,22]

The prognostic significance of the primary tumor location in GC remains debatable. In a meta-analysis of non-metastatic GC, Petrelli et al.[23] reported that tumors located in the upper third of the stomach were associated with worse survival than more distal tumors. In contrast, Nakayama et al.[24] did not observe a significant difference in survival according to tumor localization among patients with advanced GC.

In our cohort, tumor localization was associated with PFS, whereas its association with OS was not statistically significant after adjusting for other clinical variables. These

TABLE 4: Univariate and multivariate Cox regression analyses for overall survival.

	Univariate analyses		Multivariate analyses	
Variable	**HR (95% CI)**	**p-value**	**HR (95% CI)**	**p-value**
Age (per 1-year increase)	1.01 (0.99-1.02)	0.207		
Sex (male vs. female)	1.87 (1.19-2.95)	0.007	0.98 (0.53-1.82)	0.945
ECOG PS (≥1 vs. 0)	2.18 (1.33-3.55)	0.002	2.14 (1.18-3.85)	0.012
BMI (per 1 kg/m^2 increase)	1.02 (0.97-1.08)	0.358		
Tumor location (stomach vs. GEJ)	1.56 (0.98-2.48)	0.062	1.53 (0.92-2.52)	0.099
Liver metastasis (present vs. absent)	1.27 (0.85-1.89)	0.247		
Lung metastasis (present vs. absent)	1.32 (0.74-2.33)	0.347		
Peritoneal metastasis (present vs. absent)	0.85 (0.56-1.27)	0.419		
CEA status (>ULN vs. ≤ULN)	1.53 (0.99-2.36)	0.054	1.52 (0.96-2.41)	0.074
LDH status (>ULN vs. ≤ULN)	1.61 (1.02-2.53)	0.041	1.67 (0.97-2.87)	0.065
ACR group (high vs. low)	0.42 (0.28-0.63)	<0.001	0.38 (0.21-0.67)	<0.001

ACR: Albumin-to-creatinine ratio; BMI: Body mass index; CEA: Carcinoembryonic antigen; CI: Confidence interval; ECOG PS: Eastern Cooperative Oncology Group performance status; GEJ: Gastroesophageal junction; HR: Hazard ratio; LDH: Lactate dehydrogenase; ULN: Upper limit of normal.

findings suggest that the prognostic relevance of the anatomical subsite in metastatic GC is inconsistent across endpoints and may be modified by concomitant clinical and pathological characteristics. Therefore, tumor location alone may provide limited prognostic information when evaluated independently of other established risk factors.

Several aspects of this study merit further consideration. First, we evaluated a relatively homogeneous cohort of patients with *de novo* metastatic HER2-negative GC who all received first-line platinum- and fluoropyrimidine-based chemotherapy. This minimized treatment-related heterogeneity and allowed a more focused assessment of the baseline prognostic factors. Second, patients with impaired renal function (eGFR <60 mL/min/1.73 m^2) were excluded to reduce potential confounding in the interpretation of creatinine-based indices. Multivariate analyses were performed to account for established clinical and laboratory variables, thereby supporting the robustness of the association between ACR and survival outcomes.

Study Limitations

Nevertheless, several limitations of this study should be acknowledged. The retrospective, single-center design may limit the generalizability of the findings and introduce inherent selection bias. Although adequate for exploratory purposes, the sample size was modest, particularly for subgroup analyses. ACR was dichotomized according to the cohort median because no validated cutoff value currently exists. Therefore, external validation in independent populations is required. Furthermore, the ACR was assessed only at baseline, prior to treatment initiation. Temporal changes in ACR during therapy and their potential association with treatment response or survival have not been evaluated, and the prognostic implications of longitudinal ACR dynamics remain to be clarified in prospective studies. In addition, the use of immune checkpoint inhibitors in this cohort was limited, which may restrict the applicability of these findings to contemporary immunotherapy-based treatment settings. Furthermore, programmed death-ligand 1 (PD-L1) combined positive score data were not systematically available in this cohort and were therefore not included in the analysis. Accordingly, we were unable to assess whether the prognostic impact of ACR was independent of PD-L1 status.

CONCLUSION

Baseline serum ACR was independently associated with survival outcomes in patients with *de novo* metastatic HER2-negative GC. As a simple and routinely available laboratory parameter, ACR may contribute to risk stratification in advanced disease. Further prospective studies are needed to validate these findings and determine whether dynamic assessment of ACR during treatment provides additional prognostic value.

Ethics

Ethics Committee Approval: This study was conducted in accordance with the Declaration of Helsinki and approved by the İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine Ethics Committee (approval number: 1064826; date: 14.08.2024).

Informed Consent: Retrospective study.

Footnotes

Author Contributions

Surgical and Medical Practices: M.G., E.Ç., S.C., S.J., S.A., Ö.A., Concept: M.G., N.S.D., Ö.A., Design: M.G., Z.B., N.S.D., Ö.A., Data Collection or Processing: M.G., Z.B., V.A., H.A., S.C., S.J., E.Çi., Analysis or Interpretation: M.G., M.Gu., Literature Search: M.G., M.Gu., Writing: M.G., N.S.D., Ö.A.

Conflict of Interest: No conflict of interest was declared by the author.

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