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ORIGINAL ARTICLE

Reflecting, Not Defining: Limited Prognostic Value of Serum Creatinine upon Admission in Computed Tomography–Based Mortality Models for COVID-19

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What is already known on this topic?

- *Serum creatinine is commonly used as a prognostic marker in COVID-19 and is frequently elevated in patients with severe disease.*

What this study adds on this topic?

- *This study shows that while serum creatinine levels are higher in non-survivors, they do not improve mortality prediction when chest computed tomography scores and inflammatory markers are already included.*
- *The findings suggest that creatinine should be interpreted as a downstream marker of disease severity rather than an independent predictor, especially in non-kidney-specific conditions like COVID-19.*

Abstract

Objective: To challenge the perceived independent prognostic value of serum creatinine upon admission in hospitalized COVID-19 patients by evaluating its additive role in mortality prediction models that already include direct measures of pulmonary involvement and systemic inflammation.

Methods: This retrospective cohort study included 124 adults hospitalized with polymerase chain reaction–confirmed COVID-19 across 4 centers in Türkiye between March and May 2020. All patients underwent non-contrast chest computed tomography (CT) and baseline laboratory testing within 24 hours of admission. Computed tomography severity was quantified using a validated 6-zone scoring system. Four logistic regression models were constructed: model A: CT severity score; model B: CT + creatinine; model C: CT + age, diabetes, neutrophil-to-lymphocyte ratio, lactate dehydrogenase; model D: model C + creatinine. Models were evaluated by area under the curve (AUC), Brier score, accuracy, and specificity.

Results: Creatinine levels were significantly higher in non-survivors (1.12 ± 0.41 vs. 0.83 ± 0.25 mg/dL, $P = .001$). Model A yielded an AUC of 0.857; adding creatinine in model B raised the AUC modestly to 0.882. Model C, incorporating systemic and clinical variables, achieved the highest performance (AUC 0.975). Model D, which added creatinine to model C, showed no further improvement in discrimination (AUC 0.975) or calibration (Brier score 0.032).

Conclusion: Despite its widespread clinical use and association with adverse outcomes, serum creatinine offers limited incremental prognostic value when CT-based severity scoring and inflammatory markers are included. These findings suggest that creatinine reflects—but does not independently define—disease severity in COVID-19.

Keywords: Computed tomography, COVID-19, creatinine, mortality prediction

Introduction

Coronavirus disease 2019 (COVID-19), caused by the SARS-CoV-2 virus, manifests with a broad clinical spectrum ranging from asymptomatic infection to acute respiratory distress syndrome and multi-organ failure.[1] Early identification of patients at risk for severe disease or in-hospital mortality remains a critical challenge in clinical management. Several demographic characteristics—such as advanced age and pre-existing comorbidities—as well as laboratory abnormalities, including lymphopenia, elevated C-reactive protein (CRP), D-dimer, and troponin levels, have been independently associated with adverse outcomes.[2] Concurrently, radiological evaluation—particularly semi-quantitative scoring of chest computed tomography (CT) images—has emerged as a practical and reproducible method for assessing pulmonary involvement and predicting clinical trajectory in COVID-19 pneumonia.[3,4]

In a previous analysis of this same multicenter cohort, it was demonstrated that a semiquantitative CT scoring method was strongly predictive of in-hospital mortality, with a performance comparable

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to other established severity indicators (Sahutoğlu et al, 2024).[4] However, while CT scoring offers a direct assessment of the primary disease burden in the lungs, it remains unclear whether systemic biomarkers—particularly serum creatinine upon admission (Cr-UA), one of the most routinely measured and widely available laboratory tests in clinical practice—add meaningful prognostic value when imaging data are already available.

Serum creatinine is one of the most routinely measured laboratory parameters and has been widely reported as a predictor of poor outcomes in COVID-19.[5,6] Yet, its prognostic utility may be confounded by age, comorbidities, and systemic inflammation, all of which may coexist in critically ill patients.[7] Whether creatinine provides incremental value beyond established clinical and radiologic markers has not been directly evaluated in this dataset.[8]

In this secondary analysis, it was hypothesized that although Cr-UA would be elevated in non-survivors, it would not meaningfully improve predictive performance when included alongside CT severity score and other systemic indicators. To test this, 4 logistic regression models were constructed and compared to evaluate the additive role of creatinine in mortality prediction.

Methods

Study Design and Participants

This study is a secondary analysis of a previously published retrospective multicenter cohort comprising adult patients hospitalized with polymerase chain reaction (PCR)–confirmed COVID-19 across 4 hospitals in Türkiye between March and May 2020. Inclusion criteria were: age ≥18 years, a positive SARS-CoV-2 PCR test from a nasopharyngeal swab, availability of chest CT imaging and baseline laboratory tests within 24 hours of admission, and hospitalization for at least 1 day. Patients were excluded if they had severe or critical illnesses unrelated to COVID-19, typical CT findings but a negative PCR result, or were not hospitalized. Of 350 patients admitted with a provisional diagnosis of COVID-19, 124 met the criteria and were included in the analysis; 226 were excluded (166 with a negative PCR test and 60 without an admission CT scan).[4]

This analysis focuses specifically on evaluating the incremental prognostic value of Cr-UA in mortality prediction models that already include CT severity and systemic biomarkers. Ethical approval for the original study was obtained from the institutional review boards of all participating centers.

Computed Tomography Severity Scoring

Chest CT scans were assessed using a validated semi-quantitative scoring system that estimates the extent of parenchymal involvement across 6 lung zones. The scoring approach, interobserver reliability, and clinical correlation were detailed in the original study (Sahutoğlu et al, 2024).[4,9] In this secondary analysis, total CT scores were used as continuous variables in all models.

Laboratory and Clinical Variables

Baseline laboratory values—collected within 48 hours of admission—included serum creatinine, neutrophil-to-lymphocyte ratio (NLR), lactate dehydrogenase (LDH), and other standard biochemical markers. Demographic variables (age, sex), comorbidities (e.g., diabetes), and clinical outcomes (survival status) were retrieved from medical records using standardized data forms.

This study was approved by the Harran University Clinical Research Ethics Committee on April 27, 2020, with Approval No: HRU/20.08.08. Since the study is retrospective, informed consent was not required.

Statistical Analysis

Statistical analyses were performed using JASP (Version 0.19.3, University of Amsterdam) and Python (scikit-learn v1.x). Normality of continuous variables was assessed using the Shapiro–Wilk test. Comparisons between survivors and non-survivors were made using independent-samples *t*-tests for normally distributed variables (reported as mean ± standard deviation) and Mann–Whitney *U*-tests for non-normally distributed variables. Categorical variables were analyzed using Pearson's chi-square test.

To assess predictors of in-hospital mortality, 4 sequential multivariable logistic regression models were constructed based on clinical relevance and significance in univariate analysis:

- Model A: CT severity score alone
- Model B: Model A + Cr-UA
- Model C: CT severity score, age, diabetes mellitus, NLR, and LDH
- Model D: Model C + Cr-UA

Model performance was assessed using the area under the receiver operating characteristic curve (AUC), alongside model accuracy and specificity. For each model, the statistical significance of AUC > 0.5 was tested using the Hanley & McNeil method.

Pairwise comparisons of AUCs were performed using a nonparametric bootstrap approach (n = 1000 iterations). At each iteration, a bootstrap sample was drawn with replacement, models were retrained, and the AUC difference was calculated. The mean AUC difference, bias-corrected 95% CIs using the percentile method, and empirical 2-tailed *P*-values were reported for each comparison.

Although DeLong's test for correlated AUCs was initially attempted, the results were discarded due to numerical instability in the context of class imbalance and low event count. The bootstrap-based method was favored for its robustness.

All Python-based model evaluations and resampling analyses were conducted using the ChatGPT-4o model (OpenAI, 2025) in a reproducible interactive setting.

A 2-tailed *P*-value < .05 was considered statistically significant.

Results

Patient Characteristics

Baseline characteristics of the cohort, including demographic data, comorbidities, laboratory values, and CT severity scores, have been previously described and presented in Table 1. Detailed information on COVID-19–directed therapies (e.g., antivirals, corticosteroids, anti-inflammatory biologics) was not systematically available. As the study period corresponded to the early phase of the pandemic in Türkiye, there was substantial variability in management approaches across centers. The use of biologics was rare, if not absent. Briefly, the cohort consisted of 124 hospitalized COVID-19 patients, among whom 12 (9.7%) died during hospitalization. Non-survivors were significantly older and had a higher prevalence of comorbidities, as well as elevated systemic biomarkers including serum creatinine, LDH, and NLR. They also exhibited significantly higher CT severity scores at admission (Table 2).

In this secondary analysis, the focus was on the prognostic contribution of Cr-UA, particularly its performance when added to CT-based mortality prediction models.

Mortality Prediction Models and the Role of Creatinine

Four sequential logistic regression models were constructed to assess the contribution of serum creatinine to in-hospital mortality prediction (Table 3 and Figure 1).

Table 1. The Initial Clinical Characteristics and Hospitalization Details at the Start of Treatment

Variable	All Patients (n = 124)	Survivors (n = 112)	Non-Survivors (n = 12)	*P*
Age	46.13 ± 17	44.29 ± 16.2	63.3 ± 16.5	.002
Gender (M/F)	69/55	61/51	8/4	.419
Overall coexisting disorders	52/124 (41.9%)	40/112 (35.7%)	12/12 (100%)	.000
Diabetes mellitus	22/124 (17.7%)	16/112 (14.2%)	6/12 (50%)	.002
Hypertension	24/124 (19.3%)	17/112 (15.1%)	7/12 (58.3%)	.000
Coronary heart disease	3/124 (2.4%)	2/112 (1.7%)	1/12 (8.3%)	.161
Heart failure	1/124 (0.8%)	1/112 (0.9%)	0/12 (0%)	.742
Asthma	6/124 (4.8%)	6/112 (5.3%)	0/12 (0%)	.411
COPD	6/124 (4.8%)	3/112 (2.6%)	3/12 (25%)	.001
Cancer	1/124 (0.8%)	1/112 (0.9%)	0/12 (0%)	.742
CVA	2/124 (1.6%)	1/112 (0.9%)	1/12 (8.3%)	.052
Alzheimer disease	1/124 (0.8%)	1/112 (0.9%)	0/12 (0%)	.742
Seizures	1/124 (0.8%)	0/112 (0%)	1/12 (8.3%)	.002
CKD	2/124 (1.6%)	1/112 (0.9%)	1/12 (8.3%)	.052
Psychiatric disease	5/124 (4%)	4/112 (3.5%)	1/12 (8.3%)	.425
GERD	1/124 (0.8%)	1/112 (0.9%)	0/12 (0%)	.742
Hypothyroidism	1/124 (0.8%)	1/112 (0.9%)	0/12 (0%)	.742
Data of hospitalization				
Duration of hospitalization (days)	11.69 ± 8.5	11.1 ± 6.3	17.2 ± 19.6	.024
ICU admission	21/124 (16.9%)	10/112 (8.9%)	11/12 (91.6%)	.000
Oxygen support	39/124 (31.4%)	27/112 (24.1%)	12/12 (100%)	.000

COPD, chronic obstructive pulmonary disease; CKD, chronic kidney disease; CVA, cerebrovascular accident; GERD, gastroesophageal reflux disease; ICU, intensive care unit.

Table 2. Selected Baseline Laboratory and Radiologic Parameters by Survival Status*

Parameter	All Patients (n = 124)	Survivors (n = 112)	Non-Survivors (n = 12)	*P*
Creatinine (mg/dL)	0.85 ± 0.27	0.83 ± 0.25	1.10 ± 0.40	.001
LDH (U/L)	290 ± 187	264.7 ± 112.8	569 ± 455	<.001
NLR	–	2.91 ± 1.83	8.58 ± 5.72	<.001[1]
Troponin (ng/L)	40.6 ± 275	5.3 ± 12.4	465 ± 935	<.001
CRP (mg/L)	30.2 ± 45.6	25.3 ± 39.5	79.2 ± 71.0	<.001
Ferritin (ng/mL)	469.6 ± 764	343 ± 503	2212 ± 1488	<.001
CT Severity Score	15.45 ± 15.5	12.8 ± 12.0	39.8 ± 22.0	<.001

CRP, C-reactive protein; CT, computed tomography; LDH, lactate dehydrogenase; NLR, neutrophil-to-lymphocyte ratio.
[1]Calculated from original dataset; value not shown in this table in prior publication.
*This table presents only the laboratory and imaging variables used in multivariable modeling. Full baseline data for the cohort were previously reported by Sahutoglu et al, 2024.

Table 3. Logistic Regression Model Performance

Model	AUC(ChatGPT)	AUC(JASP)	Accuracy(ChatGPT)	Accuracy(JASP)	Specificity(ChatGPT)	Specificity(JASP)	Brier Score(JASP)	*P*(ChatGPT)
A	0.8571	0.857	93.5%	93.5%	99.1%	99.1%	0.059	<.001
B	0.8783	0.882	91.9%	93.5%	98.2%	99.1%	0.05	<.001
C	0.9263	0.975	95.2%	95.7%	100.0%	99.1%	0.032	<.001
D	0.9338	0.975	93.5%	95.7%	98.2%	99.1%	0.032	<.001

AUC, area under the receiver-operating-characteristic curve. Model A contains the admission chest CT severity score only. Model B adds Cr-UA. Model C further incorporates age, diabetes mellitus, neutrophil-to-lymphocyte ratio (NLR) and lactate dehydrogenase (LDH). Model D augments Model C with Cr-UA.

Model A (CT severity score alone) yielded an AUC of 0.857, with an accuracy of 93.5% and specificity of 99.1%.

Model B (CT + creatinine) showed a modest increase in AUC to 0.882; however, accuracy and specificity were unchanged.

Model C (CT + age, diabetes, NLR, LDH) achieved a higher AUC of 0.975 with improved accuracy (95.7%) and specificity (99.1%).

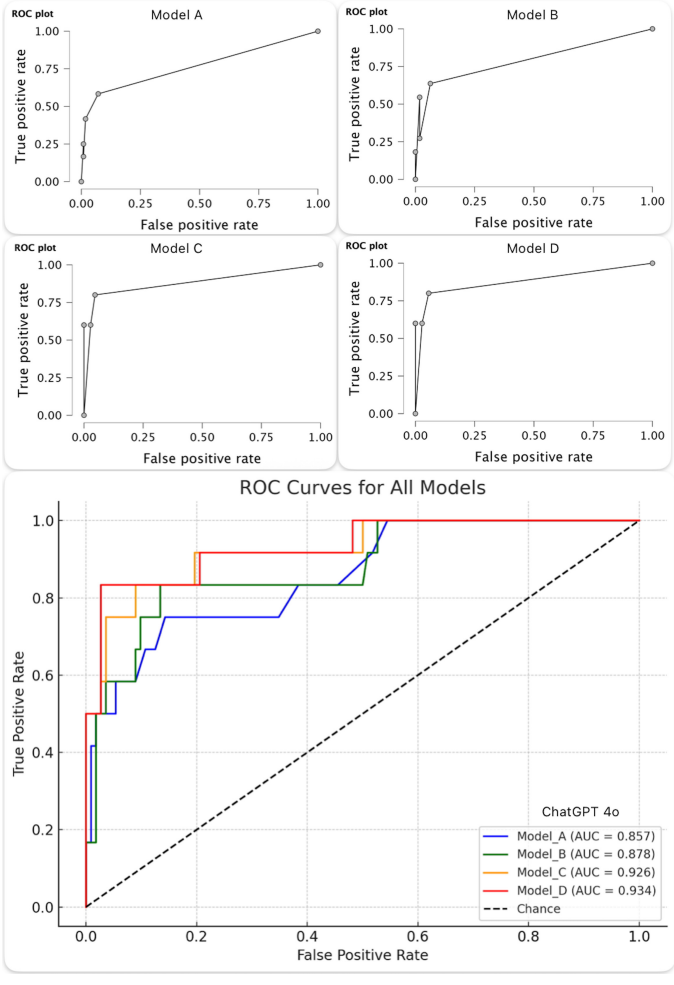

Figure 1. ROC curves comparing the discriminative performance of 4 logistic regression models for in-hospital mortality. Model A: CT score alone; Model B: CT + creatinine; Model C: CT + age, diabetes, NLR, LDH; Model D: Model C + creatinine. The addition of creatinine to either model resulted in limited or no improvement in AUC.

Model D (Model C + creatinine) did not significantly outperform Model C in terms of discrimination (Table 4). The difference in AUC between the 2 models was minimal (mean ΔAUC: −0.0072; 95% CI: [−0.040, 0.0061]; $P = .58$), confirming no added predictive value from including serum creatinine once age, CT severity score, diabetes, LDH, and NLR were already integrated. This supports the conclusion that while creatinine is elevated in nonsurvivors, it does not confer independent prognostic information beyond established severity indicators.

Brier scores, which assess model calibration, progressively declined from model A (0.059) to model B (0.050), and reached their lowest value in both models C and D (0.032). This indicates improved calibration with the inclusion of systemic clinical variables. However, since both model C and model D shared the same Brier score, and no improvement in AUC was observed, the addition of creatinine offered no incremental benefit in the best-performing model.

In contrast, the comparison between model A and model D revealed a statistically significant improvement in AUC (mean ΔAUC: −0.095; 95% CI: [−0.238, −0.0048]; $P = .028$), suggesting that creatinine may provide modest discriminatory value when added to limited models lacking broader clinical context.

These findings reinforce the principle that distal or systemic biomarkers like creatinine may lose predictive relevance in the presence of more proximate and organ-specific indicators, such as CT severity and inflammatory burden. Creatinine's initial prognostic signal appears to be absorbed by more direct covariates, limiting its utility in comprehensive models.

Discussion

In this retrospective multicenter study of hospitalized COVID-19 patients, it was found that Cr-UA levels were significantly higher among non-survivors and associated with in-hospital mortality. However, in multivariable logistic regression, Cr-UA contributed only modestly to mortality prediction. The statistical significance observed between model A (CT score alone) and model D (CT + age, diabetes, NLR, LDH + creatinine) is likely related to the greater strength of model D due to the cumulative addition of multiple strong predictors, rather than a specific independent contribution from creatinine. This interpretation is supported by the lack of significant differences between model A and model B (CT + creatinine) and between model C (CT + age, diabetes, NLR, LDH) and model D. Thus, while kidney dysfunction reflects overall disease burden, Cr-UA offers limited independent prognostic value once pulmonary involvement and systemic inflammation are already quantified.

Several studies have linked elevated serum creatinine with adverse outcomes in COVID-19, attributing its rise to systemic

Table 4. Pairwise Comparisons of Model Discrimination Using Bootstrap Resampling (1000 Iterations)

Comparison	Mean ΔAUC	95% CI	*P*
Model A vs. Model B	−0.030	[−0.106, 0.0059]	.280
Model A vs. Model C	−0.085	[−0.229, 0.0026]	.058
Model A vs. Model D	−0.095	[−0.238, −0.0048]	.028
Model B vs. Model C	−0.056	[−0.190, 0.040]	.310
Model C vs. Model D	−0.0072	[−0.040, 0.0061]	.580

hypoperfusion, cytokine-induced tubular injury, or underlying chronic kidney disease (CKD).[10-14] Cheng et al (2020)[15] and a large New York cohort both identified baseline kidney dysfunction as a mortality predictor, while Arikan et al (2021)[5] further demonstrated that advanced acute kidney injury (AKI), rather than preexisting non-dialysis CKD, was independently associated with poor outcomes in Turkish hospitalized patients.[16] The findings corroborate that non-survivors had significantly higher Cr-UA; however, its predictive value was absorbed by more proximate indicators—such as CT severity, NLR, and LDH—when included in multivariable models.[14] These results suggest that while creatinine signals global physiological stress, it functions more as a downstream correlate than a primary determinant of mortality in the acute setting.

Both the NLR and LDH have been widely studied as biomarkers of systemic inflammation and cellular injury in COVID-19.[17,18] In this cohort, these parameters were significantly elevated in non-survivors, consistent with previous findings. However, similar to serum creatinine, their predictive contributions diminished substantially in multivariable models that included chest CT severity scores. Like creatinine, NLR and LDH reflect downstream consequences of severe disease—immune dysregulation, hypoxia, and tissue damage—but do not directly measure the primary disease locus. This pattern reflects a well-known phenomenon in multivariable modeling, where distal or systemic markers lose statistical significance when more proximate and biologically central predictors are included.[19] Statistically, such variables may act as mediators or confounded correlates, with their apparent prognostic value largely explained by their association with the primary driver—in this case, pulmonary involvement as captured by CT scoring.[20]

Quantitative assessment of lung involvement via CT offers a direct, organ-specific measure of COVID-19 severity. Unlike systemic laboratory markers, CT severity scoring localizes the disease burden and correlates more closely with respiratory failure and mortality.[21] Once this central pathophysiological process is accounted for, the additional predictive value of systemic markers becomes limited. This has important implications for model development and interpretation: while markers like creatinine, NLR, and LDH may signal deterioration, their role should be understood as secondary, particularly when high-fidelity imaging is available. Prognostic models should therefore prioritize direct, disease-specific metrics to ensure both biological plausibility and predictive accuracy.[22]

This study has several limitations. First, its retrospective design limits control over confounding variables and may introduce selection bias. Second, the relatively small number of mortality events (n = 12) reduces the statistical power of multivariable modeling and increases the risk of overfitting. Third, while discrimination metrics were evaluated using robust nonparametric bootstrap methods (1000 iterations), these findings should be interpreted with caution due to the limited sample size and absence of external validation. DeLong's test was attempted but discarded due to instability in this imbalanced dataset. Fourth, although model performance appeared strong, no internal cross-validation or temporal validation was performed, limiting generalizability. Finally, creatinine's prognostic role may be confounded by age and comorbidities such as diabetes, both of which independently affect kidney function.

In summary, the findings reaffirm the primacy of chest CT severity scoring as a direct, disease-specific indicator of mortality risk in hospitalized COVID-19 patients. Although serum creatinine was significantly elevated in non-survivors, its incremental prognostic value diminished when pulmonary involvement and systemic inflammation were accounted for. This suggests that creatinine reflects downstream physiological stress rather than independently defining disease severity. While essential for assessing kidney function and identifying chronic vulnerability, its role as a predictive marker in non-kidney diseases like COVID-19 should be interpreted with caution—particularly when more proximate, organ-specific metrics are available. These findings also invite broader reflection on how creatinine and AKI are conceptualized in prognostic models of primarily non-kidney diseases, where they may serve more as consequences than cause.

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

Ethics Committee Approval: Ethical committee approval was received from the Clinical Research Ethics Committee of Harran University (Approval No: HRU/20.08.08; Date: April 27, 2020).

Informed Consent: Due to the retrospective nature of the study, informed consent is not required.

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