

Role of the Acute Physiology and Chronic Health Evaluation II (APACHE II) Score in Predicting Outcomes in Paraquat Poisoning

Praveen Kammampati ¹, Krishna Kumar M ¹, Madhusudhana Rao J.C. ¹, Venkataramanaiah E ¹

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¹. General Medicine, Sri Venkateswara Medical College and Ruia Hospital, Tirupati, IND

Corresponding author: Praveen Kammampati, kprave@gmail.com

Abstract

Background and aims

Paraquat poisoning is associated with high mortality and progressive multiorgan dysfunction. Early prognostic assessment remains difficult where toxin-specific assays are unavailable. This study evaluated the role of the Acute Physiology and Chronic Health Evaluation II (APACHE II) score in predicting mortality in patients with paraquat poisoning.

Patients and methods

This prospective observational study included 80 patients with confirmed paraquat poisoning who were admitted to a tertiary care hospital. Demographic, clinical, and laboratory data; organ dysfunction; organ support; estimated ingested dose; and APACHE II scores within the first 24 hours of ICU admission were recorded. The primary outcome was in-hospital mortality. Logistic regression was used to identify factors associated with mortality, and receiver operating characteristic (ROC) analysis assessed the discriminatory ability of APACHE II.

Results

Of 80 patients, 27 (33.8%) survived and 53 (66.2%) died. Renal dysfunction was the most frequent organ abnormality (75.0%), followed by respiratory (63.8%) and hepatic dysfunction (62.5%). Mortality increased with estimated ingested dose, from 21.1% for <20 mL to 62.5% for 20-50 mL and 91.4% for >50 mL ($p < 0.001$). PaO₂ was significantly lower among non-survivors than survivors (median 86 vs. 88 mmHg, $p = 0.034$). Median APACHE II scores were 15 in survivors and 16 in non-survivors ($p = 0.205$). APACHE II was not significantly associated with mortality on univariable analysis (OR 1.11, 95% CI 0.99-1.24; $p = 0.080$), and ROC analysis showed poor discrimination (AUC 0.587, 95% CI 0.461-0.712). In a sensitivity analysis excluding four patients with unknown or difficult-to-interpret dose estimates, recorded estimated dose showed greater discriminatory performance than APACHE II.

Conclusion

APACHE II demonstrated poor discrimination for in-hospital mortality in this cohort of patients with paraquat poisoning (AUC 0.587, 95% CI 0.461-0.712) and was not significantly associated with mortality on univariable analysis. Mortality increased with estimated paraquat dose, while lower PaO₂ was associated with mortality on univariable analysis. In the sensitivity analysis, recorded estimated dose showed greater discriminatory performance than APACHE II in the restricted patient subset. These findings indicate that APACHE II had limited discriminatory ability in the present cohort and should not be used as a stand-alone predictor of mortality. As paraquat poisoning may involve progressive organ dysfunction, a single APACHE II assessment within the first 24 hours may not fully reflect the subsequent clinical course. Future studies should evaluate whether serial APACHE II assessments provide additional prognostic information beyond the initial score. Larger prospective multicenter studies are needed to validate clinically useful prognostic models.

Categories: Internal Medicine

Keywords: apache-ii score, mortality, organ dysfunction, paraquat poisoning, prognosis

Introduction

Paraquat (1,1'-dimethyl-4,4'-bipyridinium dichloride) is a highly toxic herbicide that continues to cause severe poisoning, particularly following deliberate self-harm. Following ingestion, paraquat undergoes redox cycling and generates reactive oxygen species, resulting in cellular injury across multiple organs. The lungs are particularly vulnerable, but clinically important renal, hepatic, gastrointestinal, cardiovascular, and neurological involvement may also occur [1,2].

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The clinical course after paraquat ingestion varies substantially according to the amount and concentration of the ingested formulation, the interval between ingestion and treatment, and the extent of early organ injury. Severe poisoning may progress to acute kidney injury (AKI), respiratory failure, pulmonary fibrosis, and multiple organ dysfunction, with mortality remaining high despite advances in supportive care [2-4]. Recent studies from India have continued to report substantial mortality and frequent renal and respiratory complications among patients with acute paraquat poisoning [5-7].

Accurate early prognostication is therefore important in paraquat poisoning. Plasma or urine paraquat concentrations can provide useful prognostic information, but these investigations are not routinely available in many clinical settings. The estimated amount of paraquat ingested is another important predictor, although the history may be unreliable and the concentration of commercial formulations may vary. Other markers, including serum creatinine, arterial lactate, hematological parameters, and measures of organ dysfunction, have also been investigated as potential prognostic indicators [8-11].

Several severity scoring systems have been evaluated in paraquat poisoning. Acute Physiology and Chronic Health Evaluation II (APACHE II) is an established ICU severity score incorporating acute physiological abnormalities, age, and chronic health status. Because it is routinely available in intensive care settings, it has potential practical advantages for early risk assessment. Earlier studies have suggested that APACHE II may discriminate between survivors and non-survivors following paraquat poisoning [9,12]. Systematic reviews and meta-analyses have subsequently evaluated the prognostic performance of APACHE II, although heterogeneity among the available studies has been reported [12,13].

However, APACHE II was developed as a general severity-of-illness score and does not incorporate toxin-specific characteristics such as the amount of paraquat ingested, plasma or urine paraquat concentration, or the time elapsed since exposure. This may be particularly relevant in paraquat poisoning, in which tissue injury can progress after the initial physiological disturbance. Studies evaluating alternative prognostic approaches have demonstrated the importance of toxin burden, renal injury, hematological abnormalities, pulmonary involvement, and evolving organ dysfunction [8,10,14-17]. A 2024 prognostic nomogram study identified decreased consciousness, neutrophil-to-lymphocyte ratio, alanine aminotransferase, creatinine, carbon dioxide combining power, and plasma paraquat concentration as independent predictors of mortality [18]. Similarly, a 2026 retrospective cohort study identified ingested dose and aspartate aminotransferase (AST) as independent predictors of in-hospital mortality, with AST demonstrating an area under the curve of 0.911 [19]. Other recent studies have explored prediction models incorporating clinical and routine laboratory variables [20,21].

Accordingly, this study aimed to evaluate the predictive performance of the APACHE II score for in-hospital mortality among patients with acute paraquat poisoning and to examine its association with selected clinical and laboratory markers of disease severity.

Materials And Methods

Study design and setting

This prospective observational study was conducted in the Acute Medical Care (AMC) ICU and medical wards of Sri Venkateswara Ramnarain Ruia Government General Hospital, Tirupati, Andhra Pradesh, India. The study was conducted over a period of 12 months, from August 1, 2025, to July 31, 2026. The study protocol was approved by the Scientific and Research Committee of Sri Venkateswara Medical College, Tirupati (approval no. 311/SRC/2024, dated 09/12/2024), followed by approval from the Institutional Ethics Committee of Sri Venkateswara Medical College, Tirupati, dated 29/03/2025.

Study population

Patients aged 18 years or older who were admitted with acute paraquat poisoning during the study period were screened for eligibility. Patients of either sex were considered for inclusion. Paraquat poisoning was diagnosed on the basis of a documented history of paraquat ingestion; laboratory confirmation was not required for inclusion in the present study.

Patients were eligible for inclusion if they were aged 18 years or older, had a confirmed history of paraquat ingestion, and provided written informed consent either personally or through a legally authorized representative, when required. Patients were excluded if they had co-ingestion of another toxin or drug, pre-existing malignancy, end-stage renal disease, severe pre-existing pulmonary disease, severe pre-existing hepatic disease, or another major pre-existing condition likely to substantially affect the assessment of acute physiological severity or hospital outcome.

Data collection

All patients admitted with paraquat poisoning during the study period were assessed for eligibility according to the predefined inclusion and exclusion criteria. Eligible patients were enrolled prospectively and evaluated using a pre-structured case-record proforma. Demographic characteristics, clinical

manifestations, vital signs, Glasgow Coma Scale (GCS) [22], laboratory investigations, treatment details, and hospital outcome were recorded. Clinical assessment included gastrointestinal, abdominal, oropharyngeal, and respiratory manifestations of paraquat poisoning. Vital signs and GCS were documented during the initial clinical assessment. The history of paraquat ingestion, including the reported time of ingestion, was obtained from patients or their attendants. The times of hospital presentation and ICU admission were also documented in the case records.

Laboratory investigations included complete blood count, renal function tests, liver function tests, serum electrolytes, and arterial blood gas (ABG) analysis. Relevant clinical and laboratory parameters obtained during the initial assessment and subsequent hospital stay were recorded.

Renal dysfunction and AKI were assessed using serum creatinine and urine output. AKI was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Acute respiratory distress syndrome (ARDS) was diagnosed according to the Berlin Definition, based on the timing of onset, bilateral opacities on chest imaging, respiratory failure not fully explained by cardiac failure or fluid overload, and impaired oxygenation. The timing of organ dysfunction was determined from clinical and laboratory records at presentation and during hospitalization. Extracorporeal treatment included hemodialysis, hemoperfusion, and continuous renal replacement therapy. These modalities were used for renal support and/or attempted paraquat removal, as clinically indicated. The decision to initiate treatment and the choice of modality were made in consultation with the treating nephrologist according to the individual patient's clinical condition and treatment requirements.

The commonly available paraquat formulation in the study region was paraquat dichloride 24% soluble liquid (SL). The estimated ingested dose was based on the reported volume of formulation ingested; however, the reported volume might not accurately reflect the actual absorbed toxin burden. Patients were followed prospectively until hospital discharge or death. For the primary outcome analysis, patients were categorized as survivors or non-survivors according to their final hospital outcome.

APACHE II score assessment

The APACHE II score was used to quantify the severity of acute physiological derangement. As per the hospital protocol, all patients with acute paraquat poisoning were initially admitted to the AMC ICU and were subsequently transferred to the medical ward according to their clinical status. The APACHE II score was calculated using the most abnormal physiological values recorded during the first 24 hours following ICU admission, in accordance with the original APACHE II scoring method [23].

For the oxygenation component, APACHE II scoring was performed according to the original scoring criteria, taking into account the patient's FiO_2 and ventilatory status. The score incorporates acute physiological variables together with age and chronic health status. Acute renal failure was accounted for in APACHE II scoring according to the original scoring criteria, including the specified adjustment to the serum creatinine score when applicable.

The total APACHE II score was evaluated as the principal prognostic variable. Its association with hospital outcome was assessed by comparing scores between survivors and non-survivors and by evaluating its discriminatory performance for in-hospital mortality using receiver operating characteristic (ROC) curve analysis.

Study outcomes

The primary outcome of the study was in-hospital mortality and its association with the APACHE II score. Secondary outcomes included the clinical manifestations of acute paraquat poisoning, laboratory abnormalities, and associations between selected clinical and laboratory variables and hospital outcome. The discriminatory ability of the APACHE II score for in-hospital mortality was also assessed as part of the primary prognostic analysis.

Statistical analysis

Data were analyzed using appropriate descriptive and inferential statistical methods. Continuous variables were assessed for distribution before analysis. Normally distributed continuous variables were expressed as mean $\pm$ standard deviation, whereas non-normally distributed variables were summarized as median with interquartile range. Categorical variables were expressed as frequencies and percentages. Patients were divided into survivors and non-survivors for outcome-based comparisons. Comparisons of clinical manifestations and laboratory variables between survivors and non-survivors were considered exploratory, and the findings were interpreted cautiously in view of the multiple comparisons performed.

Normally distributed continuous variables were compared using the independent-samples t-test, while the Mann-Whitney U test was used for non-normally distributed continuous variables. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Univariable binary logistic regression analysis was performed to identify factors associated with in-hospital mortality. Results were

expressed as odds ratios (ORs) with 95% confidence intervals (CIs).

Estimated ingested dose was available for 78 patients. Two patients had unknown estimated doses; both were unconscious when found, were brought to the hospital by attendants, and subsequently died. These patients were excluded from analyses involving estimated dose.

The discriminatory ability of the APACHE II score for predicting in-hospital mortality was evaluated using ROC curve analysis. The area under the ROC curve (AUC) and its 95% confidence interval were calculated.

A sensitivity analysis was additionally performed in 76 patients after excluding the two patients with unknown estimated doses and two patients whose recorded volumes represented water-diluted mixtures rather than confirmed volumes of the original paraquat formulation. The discriminatory performance of recorded estimated dose and APACHE II was compared using ROC curves in this restricted subset, with both predictors evaluated in the same 76 patients. Confidence intervals for the AUCs and their paired difference were estimated using 10,000 stratified bootstrap resamples. An exploratory multivariable logistic regression model including recorded estimated dose and APACHE II was used to examine their independent associations with in-hospital mortality. A two-sided p-value of less than 0.05 was considered statistically significant.

Ethical considerations

The study was conducted after approval from the Institutional Ethics Committee of Sri Venkateswara Medical College, Tirupati, dated 29/03/2025. Written informed consent was obtained from all participants or their legally authorized representatives before enrolment. For patients who were unable to provide consent because of their clinical condition, consent was obtained from a legally authorized representative.

Confidentiality of participant information was maintained throughout the study. Data were anonymized for analysis and reporting, and access to study records was restricted to the investigators. All patients received standard clinical care according to the treating team's assessment. Participation in the study did not alter routine patient management. The investigations included in the study were performed without imposing an additional financial burden on the participants.

Results

The study included 80 patients with paraquat poisoning. The mean age was 33.7 ± 11.1 years, with a median age of 32 years (IQR 27–42) and a range of 18–65 years. There was a marked male predominance, with 63 (78.8%) males and 17 (21.3%) females (Table 1).

Characteristic	Value
Total patients	80
Age (years), mean ± SD	33.7 ± 11.1
Median (IQR)	32 (27–42)
Range	18–65
Gender	
Male	63 (78.8%)
Female	17 (21.3%)

TABLE 1: Baseline demographic characteristics of patients with paraquat poisoning (N = 80)

SD, standard deviation; IQR, interquartile range

Among the 80 patients with paraquat poisoning, the most common clinical manifestations were sore throat (66, 82.5%), generalized weakness (64, 80.0%), fatigue (61, 76.2%), and oral ulcers (61, 76.2%). Painful swallowing (odynophagia) and dyspnea were each present in 58 (72.5%) patients, while nausea and vomiting occurred in 55 (68.8%). Tachypnea was reported in 53 (66.2%), myalgia in 51 (63.8%), cough in 47 (58.8%), and dehydration in 45 (56.2%). Abdominal pain, esophageal ulcers/strictures, diarrhea, chest pain, and hematemesis were reported in 39 (48.8%), 34 (42.5%), 33 (41.2%), 33 (41.2%), and 15 (18.8%) patients, respectively (Table 2). Representative oral mucosal injury in paraquat poisoning, characterized by swelling, coating, ulceration, and perioral necrotic changes, is shown in Figure 1.

Clinical Characteristic	n	%
Sore throat	66	82.5
Generalized weakness	64	80.0
Fatigue	61	76.2
Oral ulcers	61	76.2
Painful swallowing (odynophagia)	58	72.5
Dyspnea	58	72.5
Nausea and vomiting	55	68.8
Tachypnea	53	66.2
Myalgia	51	63.8
Cough	47	58.8
Dehydration	45	56.2
Abdominal pain	39	48.8
Esophageal ulcers/strictures	34	42.5
Diarrhea	33	41.2
Chest pain	33	41.2
Hematemesis	15	18.8

TABLE 2: Clinical features of patients with paraquat poisoning (N = 80)



FIGURE 1: Characteristic oral mucosal injury in paraquat poisoning

Characteristic oral mucosal injury in a patient with paraquat poisoning showing a swollen, coated tongue with ulceration and perioral necrotic crusting.

Baseline laboratory evaluation demonstrated mild anemia with a mean hemoglobin concentration of 11.97 ± 2.00 g/dL. The mean total leukocyte count and platelet count were $11.34 \pm 3.17 \times 10^3/\mu\text{L}$ and $264.35 \pm 87.07 \times 10^3/\mu\text{L}$, respectively. Renal function tests showed elevated mean blood urea (70.04 ± 37.03 mg/dL) and serum creatinine (3.84 ± 2.00 mg/dL). Mean serum sodium and potassium concentrations were 137.16 ± 4.64 mEq/L and 4.05 ± 0.56 mEq/L, respectively. The mean total bilirubin was 3.92 ± 3.62 mg/dL. ABG analysis revealed a mean arterial pH of 7.30 ± 0.16 , PaO_2 of 85.24 ± 8.76 mmHg, PaCO_2 of 35.30 ± 7.80 mmHg, and HCO_3^- concentration of 20.38 ± 4.73 mEq/L. The mean APACHE II score during the first 24 hours following ICU admission was 15.92 ± 4.48 (Table 3).

Laboratory Parameter	Mean $\pm$ SD
Hemoglobin (g/dL)	11.97 $\pm$ 2.00
Total leukocyte count ($\times 10^3/\mu\text{L}$)	11.34 $\pm$ 3.17
Platelet count ($\times 10^3/\mu\text{L}$)	264.35 $\pm$ 87.07
Blood urea (mg/dL)	70.04 $\pm$ 37.03
Serum creatinine (mg/dL)	3.84 $\pm$ 2.00
Serum sodium (mEq/L)	137.16 $\pm$ 4.64
Serum potassium (mEq/L)	4.05 $\pm$ 0.56
Total bilirubin (mg/dL)	3.92 $\pm$ 3.62
Arterial pH	7.30 $\pm$ 0.16
PaO ₂ (mmHg)	85.24 $\pm$ 8.76
PaCO ₂ (mmHg)	35.30 $\pm$ 7.80
HCO ₃ ⁻ (mEq/L)	20.38 $\pm$ 4.73
APACHE II score	15.92 $\pm$ 4.48

TABLE 3: Baseline laboratory profile of patients with paraquat poisoning (N = 80)

SD, standard deviation

Comparison of clinical characteristics between survivors and non-survivors showed that sore throat (90.6% vs. 66.7%, $p = 0.008$) and oral ulcers (83.0% vs. 63.0%, $p = 0.046$) were significantly more frequent among non-survivors. Although nausea and vomiting were more common among survivors (81.5% vs. 62.3%), the difference did not reach statistical significance ($p = 0.080$). No statistically significant differences were observed for the remaining clinical characteristics (all $p > 0.05$) (Table 4).

Clinical Feature	Survivors (N = 27), n (%)	Non-Survivors (N = 53), n (%)	p-Value
Sore throat	18 (66.7)	48 (90.6)	0.008
Generalized weakness	19 (70.4)	45 (84.9)	0.124
Oral ulcers	17 (63.0)	44 (83.0)	0.046
Fatigue	18 (66.7)	43 (81.1)	0.151
Painful swallowing (odynophagia)	18 (66.7)	40 (75.5)	0.404
Dyspnea	19 (70.4)	39 (73.6)	0.761
Nausea and vomiting	22 (81.5)	33 (62.3)	0.080
Acute kidney injury	16 (59.3)	39 (73.6)	0.191
Tachypnea	20 (74.1)	33 (62.3)	0.291
Oliguria	17 (63.0)	36 (67.9)	0.657
Cough	19 (70.4)	28 (52.8)	0.132
Abdominal pain	12 (44.4)	27 (50.9)	0.582
Diarrhea	8 (29.6)	25 (47.2)	0.132
Chest pain	12 (44.4)	21 (39.6)	0.679
Altered mental status	9 (33.3)	20 (37.7)	0.699
Hematemesis	2 (7.4)	13 (24.5)	0.076

TABLE 4: Comparison of clinical characteristics between survivors and non-survivors

Pearson's chi-square test or Fisher's exact test was used, as appropriate

Baseline laboratory comparison showed that PaO₂ was significantly lower among non-survivors than survivors (median 86.00 [IQR 84.00-89.00] vs. 88.00 [IQR 85.00-91.00] mmHg, p = 0.034). No statistically significant differences were observed between survivors and non-survivors for hemoglobin, total leukocyte count, platelet count, blood urea, serum creatinine, serum sodium, serum potassium, total bilirubin, arterial pH, PaCO₂, HCO₃⁻, or APACHE II score (all p > 0.05) (Table 5).

Laboratory Parameter	Survivors (n = 27)	Non-Survivors (n = 53)	Statistical Test	p-Value
Hemoglobin (g/dL)	11.00 (10.30–12.00)	11.70 (11.10–13.00)	Mann–Whitney U	0.105
Total leukocyte count (×10 ³ /μL)	11.40 (10.40–12.25)	12.00 (9.70–13.20)	Mann–Whitney U	0.498
Platelet count (×10 ³ /μL)	250.00 (210.50–328.50)	279.00 (245.00–323.00)	Mann–Whitney U	0.390
Blood urea (mg/dL)	58.00 (45.00–78.50)	74.00 (48.00–86.00)	Mann–Whitney U	0.104
Serum creatinine (mg/dL)	2.80 (2.10–4.30)	3.60 (2.80–5.30)	Mann–Whitney U	0.114
Serum sodium (mEq/L)	137.48 ± 4.32	137.00 ± 4.82	Independent t-test	0.652
Serum potassium (mEq/L)	4.10 (3.70–4.35)	3.90 (3.70–4.26)	Mann–Whitney U	0.305
Total bilirubin (mg/dL)	1.90 (1.75–6.05)	1.90 (1.20–6.00)	Mann–Whitney U	0.733
Arterial pH	7.30 (7.20–7.35)	7.30 (7.20–7.40)	Mann–Whitney U	0.219
PaO ₂ (mmHg)	88.00 (85.00–91.00)	86.00 (84.00–89.00)	Mann–Whitney U	0.034
PaCO ₂ (mmHg)	34.00 (32.50–41.00)	34.00 (32.00–44.00)	Mann–Whitney U	0.798
HCO ₃ [−] (mEq/L)	22.00 (19.50–23.00)	22.00 (18.00–23.00)	Mann–Whitney U	0.716
APACHE II score	15.00 (14.00–16.00)	16.00 (14.00–19.00)	Mann–Whitney U	0.205

TABLE 5: Comparison of baseline laboratory parameters between survivors and non-survivors
Values are presented as median (interquartile range) for non-normally distributed variables and as mean ± standard deviation for normally distributed variables. Comparisons were performed using the Mann–Whitney U test or independent-samples t-test, as appropriate.

At presentation, renal dysfunction was the most common organ system involvement, affecting 60 (75.0%) patients, followed by respiratory dysfunction in 51 (63.8%), hepatic dysfunction in 50 (62.5%), cardiovascular dysfunction in 37 (46.3%), and neurological dysfunction in 36 (45.0%) patients (Table 6). Chest computed tomography demonstrated bilateral diffuse ground-glass opacities with interlobular septal thickening and early fibrotic changes, consistent with progressive paraquat-induced lung injury (Figure 2).

Organ System	Patients, n (%)
Respiratory dysfunction	51 (63.8%)
Renal dysfunction	60 (75.0%)
Cardiovascular dysfunction	37 (46.3%)
Neurological dysfunction	36 (45.0%)
Hepatic dysfunction	50 (62.5%)

TABLE 6: Organ System Dysfunction in Patients with Paraquat Poisoning (N = 80)

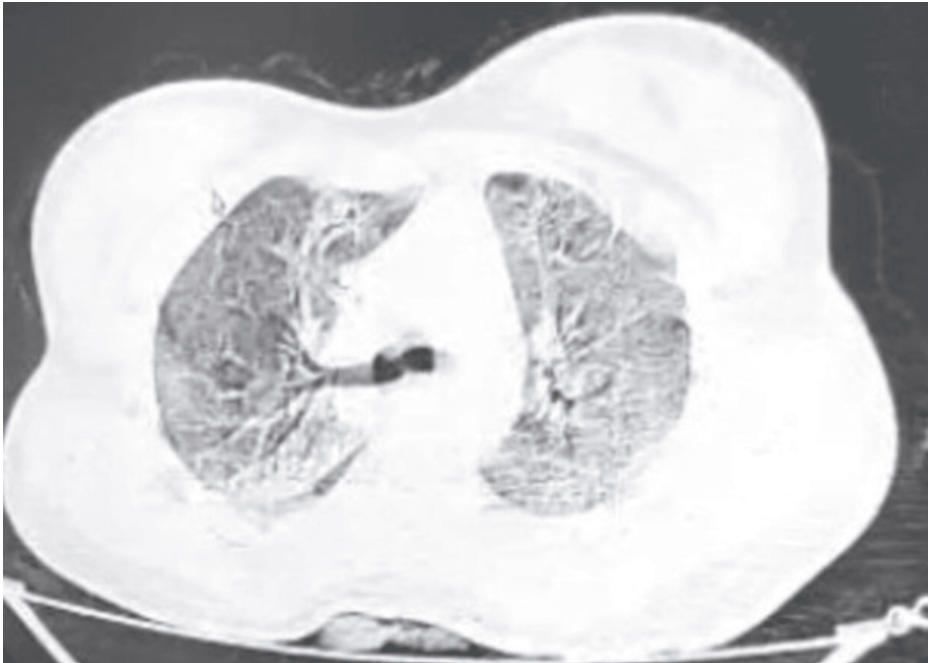


FIGURE 2: Chest computed tomography findings in paraquat poisoning

Axial computed tomography chest image showing bilateral diffuse ground-glass opacities with interlobular septal thickening and early fibrotic changes consistent with paraquat-induced lung injury.

Organ support requirements were common in both groups. Mechanical ventilation was required in 18 (66.7%) survivors and 40 (75.5%) non-survivors (p = 0.40). Dialysis support was significantly more frequent among non-survivors, being required in all 53 (100.0%) non-survivors compared with 20 (74.1%) survivors (p < 0.001). Vasopressor support was required in 17 (63.0%) survivors and 27 (50.9%) non-survivors (p = 0.31). The mean ICU stay was similar between survivors and non-survivors (8.63 ± 4.37 vs. 8.60 ± 4.32 days, p = 0.98) (Table 7).

Variable	Survivors (n = 27)	Non-Survivors (n = 53)	p-Value
Mechanical ventilation	18 (66.7%)	40 (75.5%)	0.40
Dialysis support	20 (74.1%)	53 (100.0%)	<0.001
Vasopressor support	17 (63.0%)	27 (50.9%)	0.31
ICU stay (days), mean ± SD	8.63 ± 4.37	8.60 ± 4.32	0.98

TABLE 7: Organ support and ICU outcomes among survivors and non-survivors

Chi-square test was used for categorical variables, and independent-samples t-test was used for ICU stay.

During hospitalization, ARDS was the most frequent complication, occurring in 56 (70.0%) patients. AKI and multiple organ dysfunction syndrome (MODS) each developed in 55 (68.8%) patients. Hepatotoxicity was observed in 44 (55.0%) patients, while sepsis occurred in 39 (48.8%) patients (Table 8).

Complication	Patients, n (%)
ARDS	56 (70.0)
AKI	55 (68.8)
MODS	55 (68.8)
Hepatotoxicity	44 (55.0)
Sepsis	39 (48.8)

TABLE 8: In-hospital complications of patients with paraquat poisoning (N = 80)

ARDS, acute respiratory distress syndrome; AKI, acute kidney injury; MODS, multiple organ dysfunction syndrome

Estimated ingested dose was available for 78 patients; two patients had an unknown dose. These two patients were retained as a separate “unknown” category in the descriptive dose-mortality analysis. Both patients were unconscious when found, were brought to the hospital by attendants, and subsequently died. Mortality increased progressively with increasing estimated ingested dose, from 21.1% among patients ingesting <20 mL to 62.5% among those ingesting 20–50 mL and 91.4% among those ingesting >50 mL. Dose information was unavailable in two patients, both of whom died. The association between estimated ingested dose category and mortality was statistically significant ($p < 0.001$) (Table 9).

Dose	Total	Survivors, n (%)	Non-Survivors, n (%)	Mortality Rate (%)
<20 mL	19	15 (78.9)	4 (21.1)	21.1
20–50 mL	24	9 (37.5)	15 (62.5)	62.5
>50 mL	35	3 (8.6)	32 (91.4)	91.4
Unknown	2	0 (0.0)	2 (100.0)	100.0
Total	80	27 (33.8)	53 (66.2)	66.2

TABLE 9: Mortality according to estimated ingested dose category in patients with paraquat poisoning (N = 80)

The chi-square test for the 4×2 table yielded $p < 0.001$

In univariable logistic regression analysis, platelet count (OR 1.006, 95% CI 1.001–1.012; $p = 0.030$) and PaO_2 (OR 0.91, 95% CI 0.83–1.00; $p = 0.041$) were significantly associated with mortality. Blood urea (OR 1.015, 95% CI 0.998–1.032; $p = 0.087$), serum creatinine (OR 1.29, 95% CI 0.98–1.70; $p = 0.075$), and APACHE II score (OR 1.11, 95% CI 0.99–1.24; $p = 0.080$) were not statistically significant at the 0.05 level. The remaining variables were also not significantly associated with mortality (Table 10).

Variable	Unadjusted OR	95% CI	p-Value
Age (years)	1.03	0.98–1.07	0.241
Hemoglobin (g/dL)	1.15	0.89–1.48	0.274
Total leukocyte count ($\times 10^3/\mu\text{L}$)	1.03	0.89–1.19	0.688
Platelet count ($\times 10^3/\mu\text{L}$)	1.006	1.001–1.012	0.030
Blood urea (mg/dL)	1.015	0.998–1.032	0.087
Serum creatinine (mg/dL)	1.29	0.98–1.70	0.075
Serum sodium (mEq/L)	0.98	0.88–1.08	0.659
Serum potassium (mEq/L)	0.75	0.33–1.70	0.493
Total bilirubin (mg/dL)	1.01	0.89–1.15	0.869
Arterial pH	13.18	0.50–349.37	0.123
PaO ₂ (mmHg)	0.91	0.83–1.00	0.041
PaCO ₂ (mmHg)	0.98	0.92–1.04	0.504
HCO ₃ ⁻ (mEq/L)	0.94	0.84–1.05	0.239
APACHE II score	1.11	0.99–1.24	0.080

TABLE 10: Univariable logistic regression analysis of factors associated with mortality

The discriminatory performance of the APACHE II score for in-hospital mortality was limited. ROC analysis yielded an area under the curve (AUC) of 0.587 (95% CI: 0.461–0.712), indicating poor discrimination between survivors and non-survivors. This finding was consistent with the absence of a statistically significant difference in APACHE II scores between outcome groups ($p = 0.205$; Tables 5, 11, Figure 3).

Parameter	Value
Total patients	80
Survivors	27
Non-survivors	53
AUC	0.587(95% CI: 0.461–0.712)
Interpretation	Poor discrimination

TABLE 11: Receiver operating characteristic analysis of APACHE II score for prediction of mortality

AUC, area under the receiver operating characteristic curve; APACHE II, Acute Physiology and Chronic Health Evaluation II; CI, confidence interval

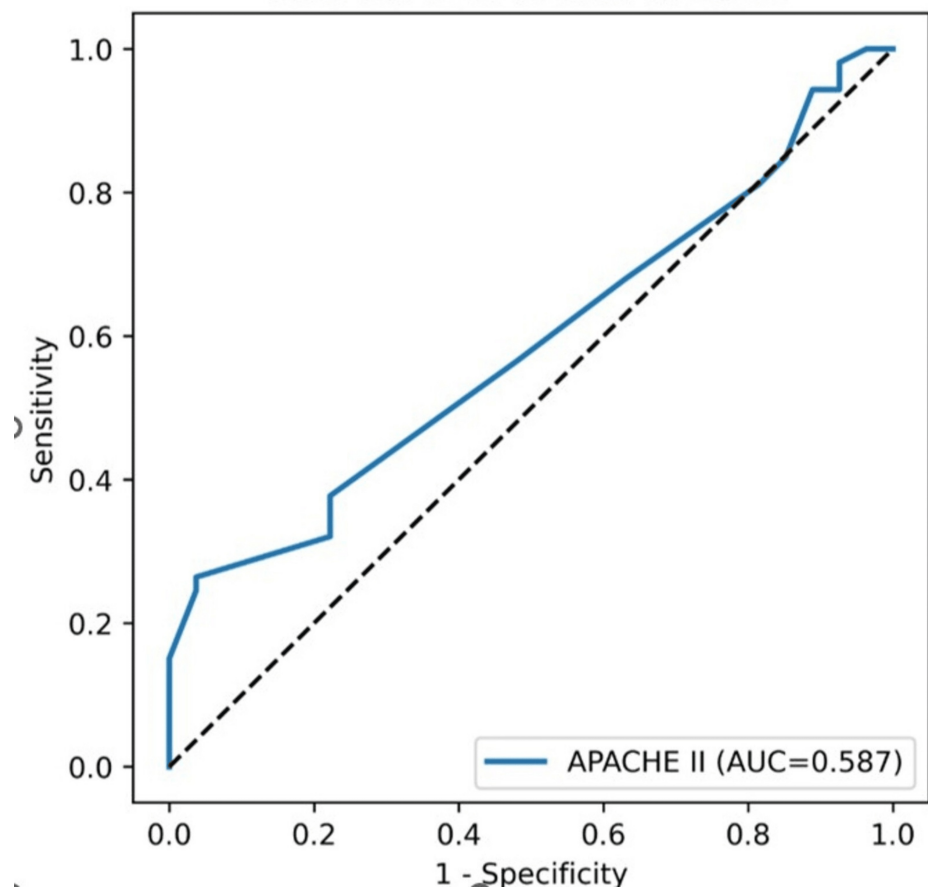


FIGURE 3: Receiver operating characteristic curve of APACHE II score for the prediction of mortality

APACHE II, Acute Physiology and Chronic Health Evaluation II; AUC, area under the receiver operating characteristic curve

Among the 27 survivors, renal dysfunction and persistent fatigue were the most common findings at hospital discharge, each occurring in 17 (63.0%) patients. Psychological symptoms were reported in 13 (48.1%), while pulmonary dysfunction persisted in 6 (22.2%) patients (Table 12).

Follow-up Finding	Survivors Affected, n (%)
Pulmonary dysfunction	6 (22.2)
Renal dysfunction	17 (63.0)
Persistent fatigue	17 (63.0)
Psychological symptoms	13 (48.1)

TABLE 12: Clinical findings among survivors at hospital discharge with paraquat poisoning (N = 27)

A sensitivity analysis was conducted in 76 patients after excluding two patients with unknown estimated doses and two patients whose recorded volumes represented water-diluted mixtures rather than confirmed volumes of the original paraquat formulation (Table 13). Mortality was 21.1% (4/19) in the lowest recorded dose category, 62.5% (15/24) in the intermediate category, and 90.9% (30/33) in the highest category (Pearson chi-square test, $p < 0.001$). The AUC was 0.858 (bootstrap 95% CI 0.756-0.940) for recorded estimated dose and 0.553 (bootstrap 95% CI 0.420-0.682) for APACHE II. The paired difference between the AUCs was 0.305 (bootstrap 95% CI 0.138-0.462), indicating greater discriminatory performance for recorded

estimated dose than for APACHE II in this sensitivity subset.

Predictor	AUC (95% CI)	Adjusted OR (95% CI)	p-Value
Recorded estimated dose, per mL	0.858 (0.756–0.940)	1.013 (1.005–1.022)	0.001
APACHE II, per point	0.553 (0.420–0.682)	1.13 (0.98–1.30)	0.092

TABLE 13: Sensitivity analysis comparing recorded estimated dose and APACHE II in 76 patients

Both ROC analyses used the same 76 patients. CIs for the AUCs and their paired difference were estimated using 10,000 stratified bootstrap resamples. The paired AUC difference (dose minus APACHE II) was 0.305 (95% CI 0.138–0.462).

AUC, area under the receiver operating characteristic curve; CI, confidence interval; OR, odds ratio; APACHE II, Acute Physiology and Chronic Health Evaluation II; ROC, receiver operating characteristic

In an exploratory multivariable logistic regression model, recorded estimated dose was associated with in-hospital mortality (adjusted OR 1.013 per mL, 95% CI 1.005-1.022; $p = 0.001$), whereas APACHE II was not independently associated with mortality after adjustment for recorded estimated dose (adjusted OR 1.13 per point, 95% CI 0.98-1.30; $p = 0.092$). These findings should be interpreted cautiously because recorded ingestion volumes were based on reported histories and may not accurately represent the volume of the original paraquat formulation or the amount absorbed. The analysis was exploratory and was based on a restricted subset of the study cohort.

ROC curves were generated to compare the performance of estimated ingested paraquat dose and APACHE II score in predicting in-hospital mortality among 76 patients. Two patients with unknown estimated doses and two patients whose recorded volumes represented water-diluted mixtures were excluded from this analysis. The AUC was 0.858 (95% bootstrap CI 0.756-0.940) for recorded estimated dose and 0.553 (95% bootstrap CI 0.420-0.682) for APACHE II. The paired AUC difference (dose minus APACHE II) was 0.305 (95% paired-bootstrap CI 0.138-0.462) (Figure 4).

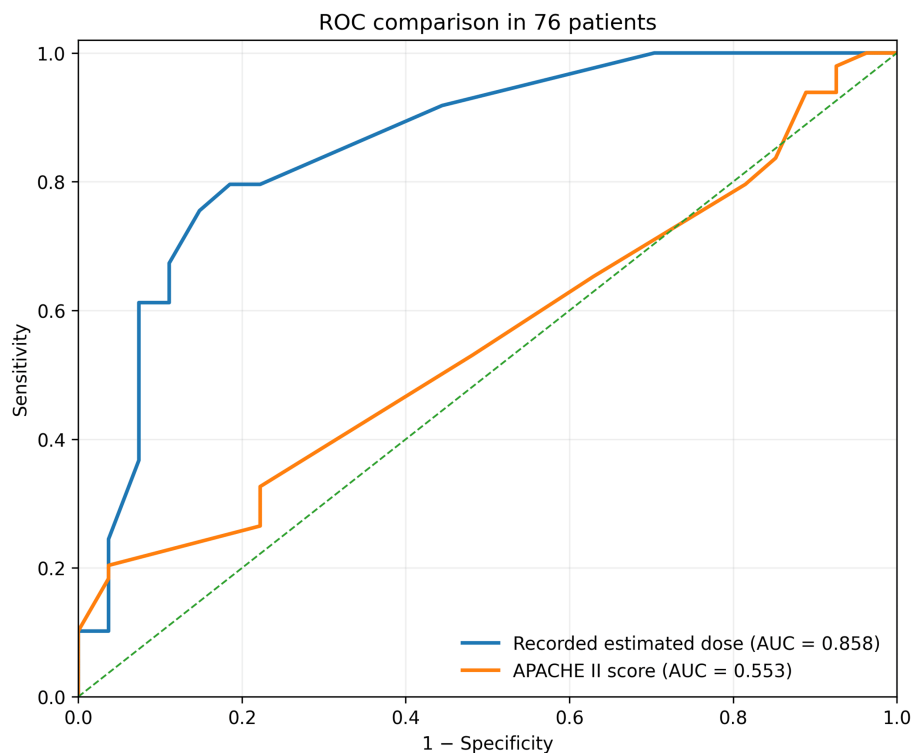


FIGURE 4: Comparison of ROC curves for recorded estimated ingested paraquat dose and APACHE II score in patients with paraquat poisoning (N = 76)

Both predictors were evaluated in the same 76 patients. The paired AUC difference (dose minus APACHE II) was 0.305 (95% paired-bootstrap CI 0.138–0.462); CIs were estimated using 10,000 stratified bootstrap resamples.

ROC, receiver operating characteristic; AUC, area under the receiver operating characteristic curve; APACHE II, Acute Physiology and Chronic Health Evaluation II; CI, confidence interval

Discussion

Paraquat poisoning remains a serious toxicological emergency because of its high case fatality and the absence of a specific antidote. In the present cohort of 80 patients, 53 (66.2%) were non-survivors. The study population was predominantly young and male, with a mean age of 33.7 ± 11.1 years and 78.8% male participants. This demographic pattern is broadly consistent with reports from Indian cohorts of acute paraquat poisoning, which have predominantly involved young adults and have reported substantial mortality [5,7].

The overall mortality in our cohort was high but within the range reported in previous studies. Ravichandran et al. reported a mortality of 72.7% in a tertiary-care cohort from South India [3], while other Indian studies have also described considerable case fatality among patients with acute paraquat poisoning [5,6]. A prospective Indian study similarly reported a substantial mortality burden and identified clinical and biochemical indicators associated with severe poisoning [7]. Differences in reported mortality between studies may reflect variation in paraquat formulation concentration, estimated dose, time to presentation, referral patterns, treatment practices, and patient selection.

The principal finding of this study was the limited ability of APACHE II to discriminate between survivors and non-survivors. The median APACHE II score was 15 among survivors and 16 among non-survivors, with no statistically significant difference between the groups ($p = 0.205$). In univariable logistic regression, APACHE II was not significantly associated with mortality (OR 1.11, 95% CI 0.99–1.24; $p = 0.080$). ROC analysis demonstrated poor discriminatory performance, with an AUC of 0.587 (95% CI: 0.461–0.712). Therefore, APACHE II showed limited ability to distinguish survivors from non-survivors in the present cohort.

This finding differs from earlier evidence suggesting a stronger prognostic role for APACHE II in paraquat poisoning. A 2017 systematic review and meta-analysis involving 29 studies reported higher APACHE II scores among non-survivors and concluded that the score had useful prognostic performance [12]. Another

systematic review and meta-analysis subsequently reported an association between APACHE II and mortality, with pooled discriminatory estimates substantially higher than that observed in the present study [13]. However, these pooled findings were derived from heterogeneous study populations, with much of the available evidence originating from East Asian centers. Differences in study design, patient characteristics, timing of APACHE II assessment, treatment practices, and outcome definitions may therefore contribute to the discrepancy between previous pooled estimates and the poor discrimination observed in our cohort.

A potential explanation is that APACHE II quantifies physiological derangement rather than the specific toxic burden produced by paraquat. Paraquat poisoning has a distinctive clinical course in which the amount absorbed, systemic distribution, and subsequent tissue injury can influence outcome [1,2]. APACHE II does not incorporate the estimated quantity of paraquat ingested, paraquat concentration, time from exposure, or toxin-specific indicators of tissue injury. Consequently, patients with substantially different toxin exposures may have similar physiological measurements during the early phase of poisoning and may therefore receive similar APACHE II scores.

The relationship between estimated toxin burden and outcome was particularly evident in our study. Mortality increased progressively from 21.1% among patients who reported ingestion of <20 mL to 62.5% among those reporting 20–50 mL and 91.4% among those reporting >50 mL ($p < 0.001$). Both patients for whom the ingested dose was unknown were non-survivors. Previous studies have also identified the amount of paraquat ingested as an important determinant of prognosis [14,19]. The dose-mortality relationship observed in our cohort therefore supports the concept that a general physiological severity score may not adequately capture the toxin-specific component of disease severity.

Renal and pulmonary involvement were prominent features of our cohort. Renal dysfunction was present in 75.0% of patients, followed by respiratory dysfunction in 63.8% and hepatic dysfunction in 62.5%. Renal injury is a well-recognized manifestation of paraquat toxicity and may reflect both direct toxic injury and the role of the kidneys in paraquat elimination. A systematic review and meta-analysis evaluating serum creatinine in paraquat poisoning reported significant prognostic value for creatinine, although heterogeneity and possible publication bias were noted [8]. Other investigations have also suggested that the evolution of renal and hepatic injury over time may provide prognostic information [15]. These findings support the importance of considering evolving organ dysfunction rather than relying solely on a single global severity score.

Pulmonary injury is another major component of paraquat toxicity. In the present study, PaO_2 was significantly lower among non-survivors than survivors, although the absolute difference was modest. Lower PaO_2 was associated with mortality in univariable analysis (OR 0.91, 95% CI 0.83–1.00; $p = 0.041$).

Nevertheless, the finding is consistent with the recognized importance of pulmonary involvement in paraquat poisoning. Previous studies have examined early pulmonary abnormalities, including CT findings and oxygenation parameters, as potential prognostic markers [16,20]. Although PaO_2 was statistically lower among non-survivors, the 2-mmHg difference in median values was small, and its clinical significance remains uncertain, particularly in the absence of a comparison adjusted for FiO_2 .

The high frequency of organ dysfunction highlights the limitations of assessing disease severity at a single time point. Paraquat toxicity is dynamic, and pulmonary, renal, hepatic, and cardiovascular abnormalities may evolve during hospitalization. APACHE II was developed primarily to quantify the severity of acute physiological disturbance rather than to capture the trajectory of toxin-mediated injury [23]. In the present study, APACHE II was assessed within the first 24 hours of ICU admission and demonstrated poor discriminatory performance, as reflected by an AUC of 0.587. A single assessment may not adequately capture the subsequent progression of physiological derangement. Serial assessment of APACHE II scores may therefore provide additional information about changes in disease severity and prognosis. Previous studies have evaluated APACHE II as a prognostic tool in paraquat poisoning [9,12,13]. These findings support further investigation of the potential value of repeated severity assessment as organ dysfunction evolves. Studies evaluating the Sequential Organ Failure Assessment score have also reported associations with mortality [11,17]. However, the present study did not evaluate serial APACHE II measurements, and their incremental prognostic value remains to be established.

Dialysis was required in all non-survivors compared with 74.1% of survivors ($p < 0.001$). This finding should not be interpreted as evidence that dialysis itself increased mortality. Rather, the markedly higher requirement for renal replacement therapy among non-survivors is likely to reflect the severity of renal and systemic illness in these patients. The substantial renal involvement observed in paraquat poisoning and the importance of renal elimination of the toxin provide a clinical context for this observation [8,15].

Recent studies have increasingly focused on prognostic models that incorporate toxin-specific and organ-specific variables. A 2024 study involving 724 patients developed a prognostic nomogram incorporating clinical and biochemical variables together with plasma paraquat concentration [18]. A more recent cohort of 128 patients identified ingested dose and AST among predictors of in-hospital death and reported strong discriminatory performance for AST [19]. Another study evaluating routine blood and urine parameters

developed a composite prognostic index incorporating several laboratory variables together with estimated dose [21]. Collectively, these studies suggest that prognostic approaches incorporating toxin burden and evolving biochemical abnormalities may provide information that is not adequately captured by a general ICU severity score alone.

The present findings should therefore not be interpreted as indicating that APACHE II has no clinical utility. APACHE II remains a practical measure of overall physiological severity and may contribute to clinical assessment when interpreted alongside other patient characteristics [23]. However, the AUC of 0.587 observed in this cohort indicates that APACHE II should not be relied upon as a stand-alone predictor of mortality in paraquat poisoning. Importantly, statistical association and predictive discrimination are distinct concepts: even when a severity score is associated with an adverse outcome, its ability to distinguish individual survivors from non-survivors may remain inadequate for clinical prediction.

Some clinical manifestations also differed between outcome groups. Sore throat and oral ulcers were significantly more frequent among non-survivors, whereas the other presenting manifestations did not differ significantly between survivors and non-survivors. These findings may reflect greater local exposure or more extensive ingestion, although the observational design of the present study does not allow a causal relationship between individual clinical manifestations and mortality to be established.

Limitations

This study has several limitations. First, the sample size was relatively small, and the study was conducted at a single tertiary-care center, which may limit the generalizability of the findings. Second, the estimated amount of paraquat ingested was based on the available history and was unavailable in two patients; consequently, the reported dose may not accurately represent the actual absorbed toxin burden. Third, plasma or urine paraquat concentrations were not available, preventing direct quantification of systemic toxin exposure. Fourth, APACHE II was assessed during the first 24 hours after ICU admission, whereas paraquat-induced organ injury may continue to evolve over subsequent days; therefore, a single early assessment may not adequately reflect the subsequent trajectory of organ dysfunction or provide useful prognostic discrimination. Larger multicenter studies incorporating standardized toxin measurements and serial assessments of organ dysfunction are therefore required to validate these findings.

Despite these limitations, the study has practical relevance because it evaluates APACHE II using routinely available clinical variables in a real-world cohort of patients with paraquat poisoning. The poor discrimination observed in our study suggests that a general ICU severity score should not be used in isolation for prognostic assessment in this toxin-specific condition. Future studies should investigate integrated prognostic models incorporating estimated dose, time from ingestion, renal and hepatic injury, lactate, oxygenation, and, where available, plasma or urine paraquat concentrations.

Conclusions

In this prospective cohort of patients with paraquat poisoning, APACHE II demonstrated poor discriminatory performance for in-hospital mortality, with an AUC of 0.587 (95% CI: 0.461-0.712). APACHE II was not significantly associated with mortality on univariable analysis. In contrast, mortality increased significantly with increasing estimated paraquat dose category, while lower PaO₂ was associated with mortality on univariable analysis. In the sensitivity analysis, recorded estimated dose showed greater discriminatory performance than APACHE II in the restricted patient subset. These findings describe the prognostic performance observed in the present cohort and should not be generalized to all patients with paraquat poisoning.

These findings suggest that APACHE II may provide information regarding general physiological severity but should not be used as a stand-alone predictor of mortality in paraquat poisoning. A single APACHE II assessment within the first 24 hours may not adequately capture the evolving severity of paraquat poisoning. Serial APACHE II assessments at different time points should therefore be evaluated in future studies to determine whether they provide additional prognostic information. Prognostic assessment may be more informative when general severity scores are integrated with toxin-specific characteristics and serial markers of evolving organ dysfunction. Larger prospective multicenter studies are required to develop and validate clinically useful prognostic models for acute paraquat poisoning.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Praveen Kammampati, Krishna Kumar M, Madhusudhana Rao J.C., Venkataramanaiah E

Acquisition, analysis, or interpretation of data: Praveen Kammampati, Krishna Kumar M, Madhusudhana Rao J.C., Venkataramanaiah E

Drafting of the manuscript: Praveen Kammampati, Krishna Kumar M, Madhusudhana Rao J.C., Venkataramanaiah E

Critical review of the manuscript for important intellectual content: Praveen Kammampati, Krishna Kumar M, Madhusudhana Rao J.C., Venkataramanaiah E

Supervision: Praveen Kammampati, Madhusudhana Rao J.C., Venkataramanaiah E

Disclosures

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