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Predictors of mortality in acute paraquat poisoning: south indian cohort analysis

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A B S T R A C T

Background: Acute paraquat poisoning is associated with high mortality, particularly in agrarian regions of South Asia. Early identification of prognostic factors is critical for guiding emergency management in resource-limited settings.

Methods: This prospective observational cohort study included 84 consecutive adults with laboratory-confirmed acute paraquat poisoning presenting to a tertiary care emergency department in South India. Demographic, exposure, clinical, and laboratory variables—including urine dithionite score and PaO_2/FiO_2 ratio—were recorded. The primary outcome was in-hospital mortality. Multivariable logistic regression was used to identify independent predictors.

Results: Overall mortality was 66.7%. Non-survivors had higher ingestion volumes, longer delays to presentation, and worse admission parameters. After adjustment, ingestion >20 mL (OR 8.45; p<0.001), urine dithionite score ≥2 (OR 6.91; p=0.001), and time to emergency department ≥4 hours (OR 4.82; p=0.004) were the strongest predictors of mortality. Reduced eGFR <75 mL/min/1.73 m² (OR 3.78; p=0.012), PaO_2/FiO_2 ratio <290 (OR 3.45; p=0.024), and CRP >50 mg/L (OR 2.98; p=0.041) were also independently associated with death.

Conclusion: Acute paraquat poisoning carries high mortality. Readily available clinical and laboratory parameters may enable early risk stratification and support timely decision-making in emergency settings, particularly in resource-limited environments.

1. Introduction

Acute paraquat poisoning remains one of the most /]]lethal toxicological emergencies encountered in emergency medicine, with a disproportionate burden in agrarian regions of South Asia (1). Paraquat, a non-selective bipyridyl herbicide, exerts toxicity through redox cycling and the generation of reactive oxygen species, leading to diffuse alveolar damage, progressive pulmonary fibrosis, acute kidney injury, and multi-organ dysfunction. Despite advances in supportive care, mortality rates remain exceedingly high, often exceeding 50–75%, with Indian studies reporting case-fatality rates approaching 60–75% even in tertiary care settings (2). The absence of a specific antidote and the widespread availability of concentrated formulations continue to contribute to its high fatality.

Epidemiological patterns in South India consistently show that paraquat poisoning predominantly affects young individuals from rural and agricultural backgrounds, with suicidal intent accounting for the majority of exposures (3). Clinical outcomes are influenced by key factors such as ingested dose, delay in presentation, and early organ dysfunction. However, early risk stratification in the emergency department remains challenging, particularly in resource-limited

settings where advanced toxicological assays are not readily available (4).

Unlike several prior regional reports that relied on retrospective analyses or examined isolated prognostic variables, the present study prospectively evaluates consecutive patients and integrates exposure characteristics, time-to-presentation metrics, toxicological severity (urine dithionite score), physiological parameters (PaO_2/FiO_2 ratio), renal function (Estimated Glomerular Filtration Rate - eGFR), and inflammatory markers (C reactive Protein - CRP) into a unified multivariable framework. By focusing on parameters readily obtainable during early emergency department evaluation, this approach seeks to bridge the gap between epidemiological observation and real-time clinical decision-making. We There fore conducted a prospective cohort study of laboratory-confirmed acute paraquat poisoning in a high-burden South Indian tertiary emergency center to identify independent predictors of in-hospital mortality and to develop a pragmatic model for early risk stratification applicable to resource-constrained settings.

2. Methodology

2.1 Study design and setting :

This was a prospective observational cohort study conducted in the Department of Emergency Medicine at a tertiary care teaching hospital in South India. The emergency department is a high-volume referral center catering to both urban and predominantly rural populations, with a significant burden of acute toxicological emergencies.

2.2 Study population:

All consecutive adult patients (≥18 years) presenting with suspected acute paraquat poisoning between December 2019 to June 2025 were screened. Patients were included if there was a definite history of ingestion and laboratory confirmation using a urine paraquat dithionite test. Only patients presenting within 24 hours of ingestion were considered to ensure assessment of early predictors. Patients with uncertain exposure, mixed or co-ingestion, chronic/occupational exposure, or those with incomplete clinical or laboratory data were excluded. Patients who were transferred after receiving definitive treatment elsewhere or who did not have a documented in-hospital outcome (e.g., left against medical advice or referred out) were also excluded.

2.3 Data collection and variables

Data were collected prospectively using a standardized case record form and included socio-demographic characteristics such as age, sex, residence (rural or urban), occupation, and intent of poisoning. Exposure-related variables comprised the type of formulation, estimated volume of paraquat ingested, calculated paraquat ion dose, use of additives during ingestion, any dilution prior to consumption, and whether the original labeled container was available at presentation. Estimated ingestion volume was obtained from patient self-report or caregiver history at presentation and corroborated, where possible, by inspection of the original container to determine formulation concentration and remaining volume. For liquid formulations, reported quantities were cross-checked against standard container sizes commonly available in the region (typically 100–250 mL). When uncertainty existed, the lower plausible estimate was recorded to minimize overestimation. Estimated paraquat ion dose was calculated based on reported volume and standard formulation concentration (20%).

Time intervals were carefully documented, including the duration from ingestion to detection, first healthcare contact, initiation of gastrointestinal decontamination, and arrival at the emergency department. Clinical parameters at admission included heart rate, systolic and diastolic blood pressure, respiratory rate, temperature, oxygen saturation, and overall time to presentation. Laboratory investigations obtained at admission encompassed serum electrolytes, complete blood counts, renal function parameters (blood urea nitrogen and estimated glomerular filtration rate), liver enzymes (aspartate aminotransferase and alanine aminotransferase), bilirubin, albumin, lactate dehydrogenase, C-reactive protein, and coagulation profile (international normalized ratio), along with arterial blood gas analysis to derive the PaO2/FiO2 ratio. Toxicological assessment was performed using a urine paraquat dithionite test, which was recorded as a semi-quantitative score ranging from 0 to 3. The variables were selected based on their availability during early emergency department evaluation, with the intent of identifying predictors that could support timely risk stratification, triage prioritization, and resource allocation in high-burden, resource-constrained settings.

All patients underwent an institutional standard treatment - supplemental oxygen was judiciously restricted and administered only when peripheral oxygen saturation fell below 85% or in the presence of overt respiratory distress. Hemodynamic stabilization was initiated with isotonic crystalloids (normal saline 20 mL/kg bolus), followed by vasopressor support when required. Norepinephrine (0.05–0.1 µg/kg/min) was used as the first-line agent, epinephrine (0.05–2 µg/kg/min) as second-line, and vasopressin (0.01–0.03 units/min) for refractory shock. Gastrointestinal decontamination was performed within 1–2 hours of ingestion when clinically appropriate. Gastric lavage was avoided due to the risk of mucosal injury; instead, activated charcoal (100 g in 100 mL water) and/or Fuller's earth (1 g/kg) were administered via nasogastric tube when available.

All patients received a standardized institutional immunosuppressive regimen consisting of intravenous methylprednisolone (1 g/day for 3 days) and cyclophosphamide (15 mg/kg/day for 2 days), followed by adjunctive antioxidant therapy with vitamin C (1 g twice daily) and vitamin E (400 mg twice daily). Renal replacement therapy (hemodialysis or hemoperfusion) was initiated early in all patients as part of the institutional protocol following hemodynamic stabilization. Metabolic acidosis (pH <7.1) was corrected using intravenous sodium bicarbonate (1–2 mEq/kg), seizures were treated with lorazepam (0.1 mg/kg IV), and electrolyte abnormalities were managed as indicated. Patients were subsequently transferred to the intensive care unit for continued monitoring and supportive care.

2.4 Outcome measure: The primary objective of this study was to identify independent predictors of in-hospital mortality in patients with acute paraquat poisoning. Secondary objectives were to compare exposure characteristics, time-to-presentation variables, admission vital signs, and laboratory parameters between survivors and non-survivors

2.5 Ethical considerations: The study was approved by the Institutional Ethics Committee (KIMS/IEC/FC010/2019-EC). Written informed consent was obtained from patients or their legally authorized representatives. The study adhered to the principles of the Declaration of Helsinki.

2.6 Statistical analysis: Statistical analysis was performed using [IBM ® SPSS Statistics for Windows, Version 26.]. All analyses were performed on a complete-case basis. During the study period, 118 patients with suspected paraquat poisoning were screened. Thirty-four were excluded due to mixed or uncertain ingestion, missing key clinical or laboratory parameters required for regression analysis, or absence of documented in-hospital outcome. The final analytic cohort therefore comprised 84 patients with complete data. No imputation procedures were undertaken.

Continuous variables were summarized as mean ± standard deviation or median with interquartile range, depending on distribution, and categorical variables as frequencies and percentages. Comparisons between survivors and non-survivors were conducted using Welch's t-test for continuous variables and chi-square or Fisher's exact test for categorical variables, as appropriate. Variables demonstrating clinical plausibility and/or an association with mortality at $p < 0.10$ in univariate analysis were considered candidate predictors for multivariable logistic regression. To preserve model stability relative to the number of outcome events, final variable selection was guided by both statistical significance and clinical relevance. To enhance bedside interpretability and facilitate pragmatic risk stratification, selected continuous variables were categorized using predefined, clinically meaningful thresholds determined a priori based on established emergency and critical care standards and the pathophysiology of paraquat toxicity. We acknowledge that dichotomization may reduce statistical power and obscure potential nonlinear relationships; however, this approach was adopted to improve clinical applicability in high-burden emergency settings. Formal sensitivity analyses modeling these predictors as continuous variables were not performed. Adjusted odds ratios with 95% confidence intervals were reported. Model adequacy was evaluated by assessing collinearity and overall fit. A two-tailed p-value < 0.05 was considered statistically significant.

3. Results

During the study period, 118 patients with suspected paraquat poisoning were screened. Of these, 9 were excluded due to mixed or uncertain ingestion, 11 were excluded because key laboratory parameters required for analysis (including urine dithionite test, renal function tests, or arterial blood gas analysis) were unavailable, and 14 were excluded due to transfer after initial stabilization (including discharge against medical advice). The remaining 84 consecutive laboratory-confirmed cases presenting within 24 hours of ingestion were included in the final analysis.

The cohort had a median age of 28 years (IQR 23.0–36.3), with a predominance of males (67.9%). Most patients were from rural areas (69.0%) and were primarily engaged in agriculture (59.5%), followed by students (23.8%). Suicidal intent was identified in the majority of cases (86.9%). Only a minority of patients were transported to the emergency department by ambulance (20.2%). (Table1)

Table 1 – Socio demographic Profile

Parameter	**Value**
Age (years), median (IQR)	28.0 (23.0-36.3)
Male gender, n (%)	57 (67.9)
Rural residence, n (%)	58 (69.0)
Occupation, n (%)	
Farmer / Agriculture	50 (59.5)
Student	20 (23.8)
Homemaker	9 (10.7)
Laborer / Other	5 (6.0)
Suicidal intent, n (%)	73 (86.9)
Transport to ED by Ambulance	17 (20.2)
ED- Emergency Medicine, IQR- Inter quartile range	

Non-survivors had significantly higher ingestion volumes, with >20 mL consumed in 53.6% compared to 17.9% of survivors (OR 9.33), and a higher mean paraquat ion dose (5.7 ± 3.1 vs 2.5 ± 1.6). Time to detection, first healthcare contact, and initiation of decontamination were significantly prolonged in non-survivors Bringing the original container was more common among survivors (71.4% vs 44.6%). No significant associations were observed for formulation type, additives, dilution prior to ingestion, or vomiting characteristics. (Table 2)

Table 2: Poison-Related Factors and Ingestion Patterns as Predictors of Mortality

Variable	*Survivors (n = 28)*	*Non-Survivors (n = 56)*	*Total (n = 84)*	*X^2 / t test OR (95% CI)*	*p-value*
Paraquat Formulation n (n%)					
Liquid concentrate (20%)	*27 (96.4)*	*54 (96.4)*	*81 (96.4)*	*1.00 (0.09-11.38)*	*0.852*
Granules / Powder	*1 (3.6)*	*2 (3.6)*	*3 (3.6)*		
Estimated Volume Ingested (mL) n (n%)					
<10 mL	*14 (50.0)*	*9 (16.1)*	*23 (27.4)*	*13.58*	*0.001*
10-20 mL	*9 (32.1)*	*17 (30.4)*	*26 (31.0)*	*2.94 (1.00-8.67)*	
>20 mL	*5 (17.9)*	*30 (53.6)*	*35 (41.7)*	*9.33 (2.89-30.12)*	
Estimated Paraquat Ion Dose (g) M±SD	*2.5 ± 1.6*	*5.7 ± 3.1*	*4.6 ± 3.0*	*5.12*	*<0.001*
Additives Used During Consumption n (n%)					
Water only	**24 (85.7)**	**50 (89.3)**	**74 (88.1)**	**0.31**	**0.855**
Alcohol	**3 (10.7)**	**4 (7.1)**	**7 (8.3)**		
Juice / Soft drink	**1 (3.6)**	**2 (3.6)**	**3 (3.6)**		
Dilution / Reconstitution Before Ingestion n (n%)					
Yes	**12 (42.9)**	**15 (26.8)**	**27 (32.1)**	**2.05 (0.78-5.40)**	**0.147**
No (ingested neat)	**16 (57.1)**	**41 (73.2)**	**57 (67.9)**		
Original Labeled Container Brought to ED n (n%)					
Yes	**20 (71.4)**	**25 (44.6)**	**45 (53.6)**	**3.10 (1.17-8.22)**	**0.023**
No / Transferred bottle	*8 (28.6)*	*31 (55.4)*	*39 (46.4)*		
Vomiting After Poison Ingestion n (n%)					
Present	*26 (92.9)*	*53 (94.6)*	*79 (94.0)*	*0.74 (0.12-4.68)*	*0.854*
Number of Vomiting Episodes (among those who vomited, n=79) M ± SD	*4.8 ± 2.9*	*6.2 ± 3.7*	*5.7 ± 3.5*	*1.89*	*0.062*
Time Interval from Ingestion to Key Events (hours), median (IQR)					
Detection by someone	*0.6 (0.3-1.0)*	*1.2 (0.6-2.5)*	*0.9 (0.4-1.8)*	-	*0.008*
First health-care contact	*2.0 (1.0-3.5)*	*4.5 (2.0-8.0)*	*3.0 (1.5-6.0)*	-	*<0.001*
Gastric lavage / decontamination	*2.5 (1.5-4.0)*	*6.0 (3.0-12.0)*	*4.0 (2.0-9.0)*	-	*<0.001*

Non-survivors demonstrated higher heart rate and respiratory rate, along with lower systolic and diastolic blood pressures and oxygen saturation at presentation (p≤0.048 for all). Time to emergency department arrival was significantly longer among non-survivors (7.4 ± 4.3 vs 3.1 ± 1.7 hours). Survivors had a longer duration of hospitalization (6.8 ± 2.1 vs 2.1 ± 1.4 days). Temperature was not significantly different between groups. (Table 3)

Table 3 – Comparison of Admission Vital Signs, Time to Presentation, and Hospitalization Duration in Acute Paraquat Poisoning

Variable	Survivors (n = 28) Mean ± SD	Non-survivors (n = 56) Mean ± SD	Mean Difference (Survivors - Non-survivors) (95% CI)	Welch's t (df)	Cohen's d	p-value
Heart Rate (bpm)	**95 ± 15**	**105 ± 18**	**-10.00 (-17.43 to -2.57)**	**-2.69 (63.7)**	**-0.59**	**0.009**
Systolic BP (mmHg)	**110 ± 12**	**100 ± 15**	**10.00 (3.96 to 16.04)**	**3.30 (65.9)**	**0.71**	**0.002**
Diastolic BP (mmHg)	**70 ± 10**	**65 ± 12**	**5.00 (0.05 to 9.95)**	**2.02 (63.7)**	**0.44**	**0.048**
Respiratory Rate (cpm)	**18 ± 3**	**20 ± 4**	**-2.00 (-3.55 to -0.45)**	**-2.57 (69.4)**	**-0.54**	**0.012**
Temperature (°C)	**37.2 ± 0.5**	**37.4 ± 0.7**	**-0.20 (-0.47 to 0.07)**	**-1.50 (71.9)**	**-0.31**	**0.137**
Oxygen Saturation (%)	**96 ± 2.5**	**94 ± 4**	**2.00 (0.58 to 3.42)**	**2.80 (77.8)**	**0.56**	**0.006**
Time to ED Arrival (hours)	**3.1 ± 1.7**	**7.4 ± 4.3**	**-4.30 (-5.61 to -2.99)**	**-6.53 (79.0)**	**-1.18**	**<0.001**
Days of Hospitalization	**6.8 ± 2.1**	**2.1 ± 1.4**	**4.70 (3.81 to 5.59)**	**10.71 (39.4)**	**2.83**	**<0.001**

BP –Blood Pressure, SD- Standard Deviation, CI- Confidence Interval, ED- Emergency Medicine

Non-survivors demonstrated significantly greater organ dysfunction, with BUN, AST, ALT, LDH, and CRP levels, along with lower eGFR and PaO_2/FiO_2 ratios ($p \leq 0.028$ for all).

Urine paraquat dithionite scores were markedly higher in non-survivors (2.13 ± 0.88 vs 1.04 ± 0.92). INR showed borderline associations, while platelet count and bilirubin were not significantly different between groups. (Table 4)

Table 4 – Comparison of Admission Laboratory and Biochemical Parameters: Survivors in Acute Paraquat Poisoning

Variable	Survivors (n = 28) Mean ± SD	Non-survivors (n = 56) Mean ± SD	Welch's t (df)	Cohen's d	p-value
Sodium (mEq/L)	139.5 ± 3.8	139.0 ± 4.3	0.51 (68.2)	0.12	0.612
Total Leukocyte Count (/mm³)	10200 ± 4800	12600 ± 7100	-1.72 (82.0)	-0.40	0.089
Neutrophil %	75.2 ± 8.4	80.0 ± 9.5	-2.41 (70.5)	-0.54	0.018
Platelet Count	218 ± 72	198 ± 80	1.17 (65.8)	0.26	0.245
BUN	22.4 ± 9.6	31.7 ± 15.4	-3.24 (82.0)	-0.70	0.002
eGFR	92.1 ± 22.4	71.5 ± 23.8	3.89 (68.4)	0.89	<0.001
AST	45 ± 28	68 ± 52	-2.68 (82.0)	-0.55	0.009
ALT	40 ± 25	58 ± 45	-2.24 (82.0)	-0.49	0.028
Total Bilirubin	0.98 ± 0.52	1.19 ± 0.74	-1.47 (78.2)	-0.32	0.145
Albumin	3.9 ± 0.5	3.7 ± 0.6	1.57 (70.1)	0.36	0.121
LDH	345 ± 140	460 ± 200	-2.85 (82.0)	-0.65	0.005
CRP	35 ± 28	72 ± 55	-3.68 (82.0)	-0.82	<0.001
INR	1.12 ± 0.18	1.21 ± 0.24	-1.85 (78.6)	-0.41	0.068
PF ratio	320 ± 68	267 ± 78	3.22 (70.9)	0.72	0.002
Urine paraquat dithionite score (0–3)	1.04 ± 0.92	2.13 ± 0.88	-5.12 (82.0)	-1.18	<0.001

BUN: Blood Urea Nitrogen (mg/dL), eGFR: Estimated Glomerular Filtration Rate (mL/min/1.73 m²), AST: Aspartate aminotransferase (IU/L), ALT : Alanine transaminase (IU/L), LDH: Lactate dehydrogenase (U/L), CRP: C-reactive protein (mg/L), INR: International Normalized Ratio, PaO2/FiO2 ratio: Arterial oxygen partial pressure (PaO2 in mmHg) to fractional inspired oxygen.

Ingestion >20 mL (OR 8.45), urine dithionite score ≥2 (OR 6.91), and time to ED ≥4 hours (OR 4.82) were the strongest independent predictors of mortality. Reduced eGFR <75 mL/min/1.73 m² (OR 3.78), PaO_2/FiO_2 <290 (OR 3.45), and CRP >50 mg/L (OR 2.98) also remained significant, while age >40 years was not (OR 2.15). (Table 5)

***Table* 5 – Multivariable Logistic Regression: Independent Predictors of Mortality in Acute Paraquat Poisoning**		
Variable	**Adjusted OR (95% CI)**	**p-value**
Ingested volume >20 mL (vs ≤20 mL)	**8.45 (2.78-25.62)**	**<0.001**
Time to ED arrival ≥4 hours (vs <4 h)	**4.82 (1.65-14.08)**	**0.004**
Urine dithionite score ≥2 (vs <2)	**6.91 (2.12-22.48)**	**0.001**
eGFR <75 mL/min/1.73 m² (vs ≥75)	**3.78 (1.34-10.65)**	**0.012**
PaO2/FiO2 ratio <290 (vs ≥290)	**3.45 (1.18-10.12)**	**0.024**
CRP >50 mg/L (vs ≤50)	**2.98 (1.05-8.47)**	**0.041**
Age >40 years (vs ≤40)	**2.15 (0.78-5.92)**	**0.138**
ED- Emergency Medicine, eGFR: Estimated Glomerular Filtration Rate (mL/min/1.73 m²), PaO2/FiO2 ratio: Arterial oxygen partial pressure (PaO2 in mmHg) to fractional inspired oxygen, CRP: C-reactive protein (mg/L).		

4. Discussion

Acute paraquat poisoning continues to impose a substantial clinical and public health burden in rural South Asia, where unrestricted access to concentrated formulations contributes to high rates of intentional ingestion (5). The 66.7% in-hospital mortality observed in this cohort is consistent with contemporary Indian series reporting mortality rates between 58% and 75% in tertiary care settings, as well as international data demonstrating wide variability depending on exposure severity and access to intensive care (1). These persistently poor outcomes, despite structured supportive care, reflect the absence of a definitive antidote and the rapid progression to multi-organ dysfunction, particularly progressive respiratory failure secondary to pulmonary toxicity.

In this study, ingested volume emerged as the strongest independent predictor of mortality, with ingestion >20 mL conferring markedly increased risk. This finding reinforces the well-established dose response relationship in paraquat toxicity, wherein higher systemic exposure overwhelms endogenous antioxidant defenses, amplifies redox cycling, and accelerates cellular injury (1,6). Similar associations between ingested dose and mortality have been reported in prior toxicological studies, including analyses by Dinis-Oliveira et al., highlighting the prognostic relevance of exposure magnitude (7). Although estimation of ingested volume is inherently imprecise, it remains a pragmatic and immediately available parameter for early risk assessment in emergency settings.

Time to presentation was another critical determinant of outcome. Patients presenting ≥4 hours after ingestion demonstrated significantly higher mortality, with non-survivors exhibiting delays across detection, first healthcare contact, and emergency department arrival. Comparable findings have been reported in Indian cohorts, emphasizing that delayed presentation substantially worsens prognosis (8). This is biologically plausible, as paraquat is rapidly absorbed and redistributed into target organs particularly the lungs and kidneys, within hours of ingestion. Early gastrointestinal decontamination using adsorbents such as activated charcoal or Fuller's earth is most effective within the first 2–4 hours, beyond which systemic toxicity predominates (9). These observations underscore the importance of rapid recognition, early referral, and streamlined prehospital care pathways.

The urine sodium dithionite test demonstrated robust prognostic utility in this cohort. Higher semi-quantitative scores (≥2) independently predicted mortality, consistent with prior evidence that greater color intensity correlates with systemic paraquat burden and clinical severity (10). Plasma paraquat concentration has traditionally been regarded as a reference prognostic marker, with earlier studies reporting that survivors rarely exceeded concentrations of approximately 2.6 µg/mL at 3 hours, whereas levels above 3.4 µg/mL were uniformly fatal. However, those data also demonstrated considerable overlap at lower concentrations, with deaths occurring even at minimal plasma levels later in the clinical course, limiting its reliability in certain time windows (11). In many resource-constrained emergency settings where quantitative plasma assays are unavailable, bedside urine dithionite testing provides a practical surrogate marker of systemic exposure. While not a direct substitute for plasma quantification, it offers immediate, clinically actionable information for early risk stratification and triage in high-burden environments (10).

Markers of early organ dysfunction further refined prognostication. Reduced eGFR (<75 mL/min/1.73 m²) likely reflects both direct nephrotoxicity and impaired toxin clearance, leading to sustained systemic exposure. Prior studies have demonstrated that paraquat is predominantly renally eliminated, and even modest declines in renal function can significantly increase circulating toxin levels (12). Similarly, a PaO_2/FiO_2 ratio <290 indicates early pulmonary

involvement, consistent with paraquat's preferential uptake into alveolar cells via polyamine transport systems, resulting in oxidative lung injury and progressive fibrosis. Pulmonary involvement has consistently been identified as the principal determinant of mortality, with extremely high fatality once significant respiratory compromise develops (13). Elevated CRP (>50 mg/L) reflects an amplified inflammatory response, which may further exacerbate oxidative tissue damage (14).

Differences in admission physiology in our cohort is characterized by tachycardia, lower blood pressure, and reduced oxygen saturation among non-survivors likely reflect early hemodynamic instability and evolving multi-organ dysfunction. Similar patterns have been reported in prior studies, where abnormal vital parameters at presentation were associated with increased mortality; tachycardia and hypotension have been observed in approximately 50–70% of severe paraquat poisoning cases and correlate with poor outcomes (15). Survivors demonstrated longer hospitalization, consistent with earlier reports where survivors required prolonged supportive care (mean 5–10 days), whereas non-survivors typically succumbed early, often within 48–72 hours of admission due to rapid progression of systemic toxicity (9).

Variables such as formulation type, co-ingestants, dilution, and vomiting were not associated with mortality, indicating limited prognostic relevance. This is consistent with prior studies in which the majority of exposures (>90%) involve liquid paraquat formulations, reducing variability in exposure characteristics (16). Although vomiting is reported in up to 80–95% of cases, its association with outcomes has been inconsistent across studies (3,16). The observed association between presentation with the original container and improved survival may reflect earlier healthcare access or more accurate exposure identification, but has not been consistently reported and should be interpreted with caution.

Despite multiple therapeutic strategies, no specific antidote exists for paraquat poisoning, and management remains largely supportive (1,9). Interventions such as hemodialysis and hemoperfusion may offer benefit when initiated early, but their effectiveness diminishes once significant tissue distribution has occurred (17,18). Similarly, the role of antioxidants and immunosuppressive therapies, including corticosteroids and cyclophosphamide, remains controversial, with inconsistent evidence regarding survival benefit across studies (19, 20). These findings reinforce that early identification of high-risk patients and timely intervention are more impactful than late-stage therapies.

Importantly, this study adds to existing literature by integrating readily available clinical, biochemical, and toxicological parameters into a unified, bedside-applicable prognostic framework. The combined assessment of ingestion volume, presentation delay, urine dithionite score, renal function, oxygenation status, and inflammatory markers provides a pragmatic approach to early risk stratification in high-volume emergency settings. These variables may also form the basis of a clinically applicable risk prediction model, warranting external validation.

From a clinical perspective, these findings enable early identification of high-risk patients at presentation, facilitating prioritization of decontamination, close monitoring, and timely escalation of care, including appropriate allocation of intensive care resources. From a public health standpoint, the predominance of intentional ingestion among young rural individuals highlights the urgent need for regulatory control of paraquat availability, safer storage practices, and strengthened mental health interventions (21). Experience from countries where paraquat use has been restricted or banned has demonstrated reductions in poisoning-related mortality, supporting the role of regulatory interventions (22,23).

This study has few limitations. Estimation of ingested volume was based primarily on patient or caregiver reporting and is inherently susceptible to recall bias. Although container verification and formulation concentration were used to support exposure estimation when feasible, misclassification cannot be excluded. Underestimation could attenuate the observed dose response association, whereas overestimation might exaggerate effect sizes. However, the persistence of ingestion volume as an independent predictor in adjusted analysis suggests that the observed association is directionally robust. Plasma paraquat concentration, a reference standard for toxicokinetic prognostication in established concentration time models, was not available. This can limit direct comparison with plasma based survival nomograms and restricts precise exposure outcome correlation. Risk stratification therefore relied on early clinical, biochemical, and bedside toxicological parameters, which may reduce alignment with concentration based models but actually reflect real world practice in resource constrained emergency settings. Selection bias cannot be entirely excluded. The analytic cohort comprised patients with complete outcome and predictor data, and exclusion of certain cases may have influenced mortality estimates. The direction of this potential bias is uncertain, as excluded patients could have differed in either severity or clinical trajectory. Additionally, as a single center study conducted in a high volume tertiary emergency department serving a predominantly rural population, the findings may not be directly

generalizable to other healthcare systems or geographic contexts

5. Conclusion

Acute paraquat poisoning remains associated with high mortality in resource-limited settings. In this cohort, ingestion volume, delay in presentation, urine dithionite score, renal dysfunction, impaired oxygenation, and systemic inflammation were key

predictors of outcome. These readily available parameters may support early risk stratification and clinical decision-making. Broader preventive strategies, including regulation of access and improved awareness, remain essential to reduce disease burden.

Declarations

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