Section: General Medicine

Original Research Article

A STUDY OF HAEMATOLOGICAL INDICES, ASSESSMENT SCORES AND PREDICTOR OF PROGNOSIS IN PATIENTS WITH ACUTE PARAQUAT POISONING

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Received : 17/04/2025
Received in revised form : 03/06/2025
Accepted : 24/06/2025

Keywords:
Paraquat poisoning, Sequential Organ Failure Assessment (SOFA), Acute Kidney Injury Network (AKIN), haematological indices, neutrophil-to-lymphocyte ratio (NLR).

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DOI: 10.47009/jamp.2025.7.3.199

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2025; 7 (3); 1030-1036

ABSTRACT

Background: Paraquat poisoning is a significant public health concern in many developing countries because of its high toxicity and lack of a specific antidote. Acute paraquat ingestion leads to multi-organ failure, with pulmonary, renal, and hepatic complications being the most common. This study aimed to evaluate the haematological indices, assessment scores, and prognostic predictors in patients with acute paraquat poisoning. **Material and Methods:** This observational study included 50 patients with acute paraquat poisoning who were admitted to the Poison Centre, Rajiv Gandhi Government General Hospital, Chennai, between August 2018 and July 2019. Clinical parameters, laboratory investigations, Sequential Organ Failure Assessment (SOFA) scores, and Acute Kidney Injury Network (AKIN) scores were recorded. **Result:** Of 50 patients, 54% were male, and 77.8% were aged 20–50 years. Ingestion was the primary route, with 62% of patients consuming 20–50 mL of paraquat. Acute respiratory distress syndrome (ARDS) developed in 38% of cases, and mortality was highest between 3- and 7 days post-ingestion (28%). The mean arterial blood gas (ABG) pH declined from 7.21±0.22 at admission to 6.99±0.33 at 48 h (p=0.002), while leucocyte counts increased to 17,473±28,566 cells/µL at 24 h. The neutrophil-to-lymphocyte ratio (NLR) increased from 3.96 ± 1.67 at admission to 4.18±1.87 at 24 hours. Higher SOFA and AKIN scores were associated with ARDS, mechanical ventilation (p=0.006), and haemodialysis (p=0.003). **Conclusion:** Leucocytosis, neutrophilia, and elevated NLR are strong prognostic markers of acute paraquat poisoning. SOFA and AKIN scores at 48 h effectively predicted disease progression, emphasising the need for early intensive management, including pulse therapy.

INTRODUCTION

Paraquat poisoning is a significant health problem, particularly in developing countries, where the herbicide remains widely used despite its toxicity. Paraquat is a highly toxic bipyridyl herbicide that is lethal when ingested, even in small quantities. As little as one teaspoon of paraquat can be fatal.[1] Once absorbed into the body, paraquat undergoes redox cycling, generating reactive oxygen species (ROS) that cause widespread cellular damage. This results in multi-organ failure, with the lungs, kidneys, liver, and heart being most affected.[2] Due to the rapid progression of toxicity and lack of an effective antidote, mortality remains high, often occurring within hours to days after ingestion.[3]

Paraquat toxicity is primarily due to its ability to generate free radicals, which induce oxidative stress and lipid peroxidation, leading to cellular apoptosis and necrosis. This oxidative damage predominantly affects the alveolar epithelium in the lungs, leading to progressive pulmonary fibrosis, a major cause of death in paraquat poisoning. Additionally, acute kidney injury (AKI) and hepatotoxicity frequently complicate the clinical course, further contributing to poor patient outcomes.[4] The severity of poisoning is influenced by the dose ingested, the patient's metabolic status, and the time to hospitalisation after ingestion. Given the challenges in managing paraquat poisoning, early risk stratification using clinical and laboratory parameters is crucial for guiding treatment decisions.[5]

Recent studies suggest that haematological indices, such as leukocytosis, neutrophilia, and the

ISSN (O): 2687-5365; ISSN (P): 2753-6556

neutrophil-to-lymphocyte ratio (NLR), may serve as potential prognostic markers.[4] Furthermore, scoring systems such as the Sequential Organ Failure Assessment (SOFA) and the Acute Kidney Injury Network (AKIN) classification have been utilized to predict disease progression and mortality risk.[6] Understanding the prognostic significance of these markers can facilitate early intervention strategies, including aggressive supportive therapy and immunosuppressive treatments such as pulse therapy with methylprednisolone and cyclophosphamide.[7]

Despite its high toxicity, paraquat remains widely used in agricultural settings because of its effectiveness as a non-selective herbicide. In many countries, restrictions have been placed on its sale and use; however, illegal access and accidental or intentional ingestion continue to contribute to poisoning cases.[8] The World Health Organization (WHO) classifies paraquat as a moderately hazardous substance, whereas the United States classifies it as highly dangerous. There is an urgent need for public health interventions to regulate its availability and promote safer alternatives.[9]

Paraquat poisoning remains a major public health concern, with high morbidity and mortality due to the lack of a specific antidote and limited treatment options. Early identification of high-risk patients is crucial for optimising management and improving survival rates. Studies have highlighted the prognostic value of haematological indices and organ failure scores; however, validation is needed in different populations and settings. Hence, this study aimed to investigate the haematological indices and assessment scores and evaluate the predictors of prognosis in patients with acute paraquat poisoning.

MATERIALS AND METHODS

This observational study included 50 patients from the poison centre, Institute of Internal Medicine, Rajiv Gandhi Government General Hospital, Chennai, from August 2018 to July 2018. The Institutional Ethics Committee approved this study before it began, and all patients provided informed consent for participation.

Inclusion Criteria

All patients with acute paraquat poisoning admitted to our hospital, irrespective of age and sex, with a positive urine sodium dithionite test were included.

Exclusion Criteria

Patients with a history of pesticide poisoning, chronic liver disease, malignancy, chronic kidney disease, previous pulmonary disorders, or a negative urine sodium dithionite test result were excluded.

Methods

Patients admitted to the emergency department with a history of paraquat ingestion were screened using the urine dithionite test. Routine investigations included arterial blood gas (ABG) analysis, renal function test (RFT), liver function test (LFT), C-reactive protein (CRP) levels, complete blood count (CBC), random blood sugar (RBS) level, and urine sodium dithionite test. All laboratory investigations were performed according to standard protocols. Sequential Organ Failure Assessment (SOFA) score and AKI staging were compared.

Statistical Analysis

Data are presented as mean, standard deviation, frequency, and percentage. Continuous variables were compared using the independent sample t-test and ANOVA. Categorical variables were compared using Pearson's chi-square test. Significance was defined as $p < 0.05$ using a two-tailed test. Data analysis was performed using IBM SPSS version 21.0.

RESULTS

Of the 50 patients, 43.55% were male, and those aged 20–50 years comprised 77.8% of the patients. Males constituted 54%, with the majority (77.8%) falling within the 20–50 years age group. Ingestion was the primary route of exposure, with 62% consuming 20–50 mL of paraquat and 12% consuming <20 mL. The most commonly consumed formulation was AVAST 24% (30%), followed by SWAT 24% and KAPIQ 24% (28% each). A significant proportion (76%) of patients presented to the hospital within 6–24 h of ingestion. The urine sodium dithionite test revealed that 40% of patients exhibited a light blue reaction, 36% showed a dark blue response, and 24% had a barely distinguishable blue colour. The duration of hospitalisation varied, with 28% of patients requiring a stay of > 15 days. ARDS was the most common complication, occurring in 38% of patients. Mortality was highest between 3- and 7-days post-ingestion (28%) (Table 1).

Table 1: Clinical characteristics and outcomes

		N (%)
Sex	Male	27 (54%)
	Female	23 (46%)
Age (years)	< 20	11 (22%
	20-50	34 (68%)
	> 50	5 (10%)
Amount of consumption of PQ (ml)	< 20	6 (12%)
	20-50	31 (62%)
	50-150	7 (14%)
	>150	6 (12%)
Frequency of substance used	HERAQUAT 24%	7 (14%)
	AVAST 24%	15 (30%)

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	SWAT 24%	14 (28%)
	KAPIQ 24%	14 (28%)
Time between PQ Ingestion and hospital arrival (hours)	< 6	4 (8%)
	Jun-24	38 (76%)
	> 24	8 (16%)
Urine sodium dithionite test	Barely Distinguishable Blue (1+)	12 (24%)
	Light Blue (2+)	20 (40%)
	Dark Blue (3+)	18 (36%)
	Black (4+)	0
Duration of hospital stay	24 hours	5 (10%)
	48 hours	13 (26%)
	7 Days	14 (28%)
	8-15 days	10 (20%)
	> 15 days	8 (16%)
Complications of PQ poisoning	Metabolic Acidosis	6 (12%)
	AKI	13 (26%)
	ARDS	19 (38%)
	MODS	12 (24%)
Mortality	< 24 hours	5 (10%)
	48 hours	13 (26%)
	3-7 days	14 (28%)
	8-14 days	10 (20%)
	> 15 days	8 (16%)

The ABG parameters showed significant alterations over time. The mean pH at admission was 7.21±0.22, which declined to 7.10±0.25 at 24 h and further decreased to 6.99±0.33 at 48 h (p=0.002). PaO_2 levels showed a significant reduction from 92.06±38.13 mmHg on admission to 139.77±50.31 mmHg at 48 h (p<0.001). The PaO_2/FiO_2 ratio also showed a significant decline from 306.00±88.36 on admission to 244.75±133.00 at 48 h (p=0.026), and bicarbonate levels did not show a significant change over time (p=0.051) (Table 2).

Table 2: Comparison of ABG analysis on admission to 48 hours

ABG Analysis	Time interval	Mean±SD	P-value
pH	On Admission	7.21±0.22	0.002
	24 hours	7.10±0.25	
	48 hours	6.99±0.33	
PaO_2	On Admission	92.06±38.13	<0.001
	24 hours	201.20±102.80	
	48 hours	139.77±50.31	
$PaCO_2$	On Admission	46.63±3.97	0.004
	24 hours	44.92±3.79	
	48 hours	43.63±4.45	
HCO_3	On Admission	21.59±3.16	0.051
	24 hours	19.81±4.54	
	48 hours	19.59±4.99	
PaO_2/FiO_2	On Admission	306.00±88.36	0.026
	24 hours	258.83 ± 110.40	
	48 hours	244.75 ± 133.00	

Leukocytes were observed throughout hospitalisation, with mean leukocyte counts rising from 11,842±5,640 cells/µL at admission to 17,473.11±28,565.83 cells/µL at 24 h before decreasing to 11,898±6,062.66/mm^3 at 48 h. Neutrophil and lymphocyte count showed no significant changes. However, the neutrophil-to-lymphocyte ratio (NLR) increased from 3.96±1.67 at admission to 4.18±1.87 at 24 h, before decreasing slightly to 3.79±2.05 at 48 h.

Serum creatinine levels increased from 3.21±2.19 mg/dL at admission to 4.22±2.6 mg/dL at 24 h and remained higher at 48 h (4.05±2.97 mg/dL). Serum bilirubin levels were also elevated, increasing from 3.49 mg/dL at admission to 4.25 mg/dL at 24 h, before decreasing to 3.79 mg/dL at 48 h. CRP levels progressively increased from 5.97±2.42 mg/dL at admission to 7.57±2.96 mg/dL at 24 h and 7.32±3.38 mg/dL at 48 h (Table 3).

Table 3: Haematological and biochemical parameters

Haematological Parameters	Time interval	Mean±SD
Leukocyte (cells/µL)	On Admission	11842±5640.35
	24 hours	17473.11±28565.83
	48 hours	11898±6062.66
Neutrophil (cells/µL)	On Admission	4758.6±940.26
	24 hours	4907.11±1264.96
	48 hours	4683.03±1024.03
Lymphocyte (cells/µL)	On Admission	1390.2±523.37

ISSN (O): 2687-5365; ISSN (P): 2753-6556

	24 hours	1385.82±580.6
	48 hours	1582.33±719.02
Platelet (PL/µL)	On Admission	111840±38261.91
	24 hours	102845.33±51208.19
	48 hours	112666.67±50800.71
Haemoglobin (g/dL)	On Admission	9.16±0.67
	24 hours	9.00±0.51
	48 hours	8.42±2.1
Neutrophil-Lymphocyte Ratio (NLR)	On Admission	3.96±1.67
	24 hours	4.18±1.87
	48 hours	3.79±2.05
Urea (mg/dL)	On Admission	81.6±41.02
	24 hours	93.47±40.9
	48 hours	86.43±51.23
Serum Creatinine (mg/dL)	On Admission	3.21±2.19
	24 hours	4.22±2.6
	48 hours	4.05±2.97
Serum Bilirubin (mg/dL)	On Admission	3.49
	24 hours	4.25
	48 hours	3.79
CRP (mg/dL)	On Admission	5.97±2.42
	24 hours	7.57±2.96
	48 hours	7.32±3.38
Hypotension	On Admission	74.82±16.14
	24 hours	67.8±16.28
	48 hours	72.33±16.48

The SOFA scores increased significantly over time. At admission, 60% of patients had scores between 7 and 12, whereas at 24 h, 64% had scores between 13 and 24, indicating progressive organ dysfunction. Similarly, AKI progression was evident, with 54% of patients presenting with stage 1 AKI at admission, which increased to 26.7% with stage 3 AKI at 48 h (Table 4).

Table 4: Comparison of SOFA score and AKI staging with time interval

		Time interval		
		On admission (n=50)	**24 hours (n=45)**	**48 hours (n=32)**
SOFA Score	0-6	16 (32%)	14 (31%)	13 (41%)
	7-12	30 (60%)	2 (4%)	9 (28%)
	13-24	4 (8%)	29 (64%)	10 (31%)
AKI stage	0	15 (30%)	14 (31.1%)	11 (34%)
	1	27 (54%)	9 (20%)	3 (9%)
	2	4 (8%)	10 (22.2%)	6 (19%)
	3	4 (8%)	12 (26.7%)	12 (38%)

A significant association was found between SOFA scores and the need for mechanical ventilation (p=0.009 at admission, p=0.02 at 24 h, and p=0.006 at 48 h). SOFA scores also correlated significantly with haemodialysis requirements, with a mean SOFA score of 12.3±1.57 at 48 h in those requiring haemodialysis (p=0.003). Pulse therapy was associated with significantly higher SOFA scores at 48 h (11.375±3.36 vs. 6.44±4.41, p=0.001) (Table 5).

Table 5: SOFA scores with mechanical ventilation, haemodialysis, and pulse therapy

			SOFA score (Mean±SD)	**P-value**
Mechanical ventilation	On admission	Yes	11.375±1.59799	0.009
		No	7.6905±3.73179	
	24 hours	Yes	17.3333±2.73861	0.02
		No	11.7778±6.72781	
	48 hours	Yes	11.6667±2.80692	0.006
		No	7.25±4.72257	
Haemodialysis	On admission	Yes	11.375±1.59799	0.009
		No	7.6905±3.73179	
	24 hours	Yes	14.8889±5.13224	0.093
		No	11.5556±7.06744	
	48 hours	Yes	12.3±1.56702	0.003
		No	7.3636±4.71619	
Pulse therapy	On admission	Yes	10.4444±2.06828	0.009
		No	7.8049±3.85499	
	24 hours	Yes	15.3±6.65081	0.187
		No	12.2±6.40221	
	48 hours	Yes	11.375±3.36403	0.001
		No	6.4375±4.41163	

ISSN (O): 2687-5365; ISSN (P): 2753-6556

SOFA scores were significantly correlated with the volume of paraquat ingested. Patients consuming ≤20 mL had the lowest SOFA scores at admission (2.67±1.03), while those ingesting >150 mL had the highest (10.83±2.4, p=9.311). This continued at 24 h, with scores higher at 17.86±1.77 in the 51–150 mL group.

At 48 h, the 21–50 mL group had higher scores (9.96±4.22), but data for higher ingestion groups were unavailable, likely due to early mortality (Table 6).

Table 6: SOFA scores at various intervals based on paraquat ingestion amount

SOFA Score	Volume of paraquat ingested	Mean±SD	P-value
Admission (ml)	Up to 20	2.67±1.03	9.311
	21-50	8.42±3.52	
	51-150	10.29±1.7	
	Above 150	10.83±2.4	
24 hours (ml)	Up to 20	3.17±0.41	16.741
	21-50	13.65±6.03	
	51-150	17.86±1.77	
	Above 150	2.17±5.31	
48 hours (ml)	Up to 20	3.2±0.45	12.482
	21-50	9.96±4.22	
	51-150	-	
	Above 150	-	

Patients who developed ARDS had significantly higher SOFA scores (17.2±5.6 vs. 3.1±0.5, p<0.001), AKI scores (19±3.34 vs. 13±2.26, p<0.001), leukocyte counts (16,000±3,901.12 vs. 6,533.85±3,677.71, p<0.001), and neutrophil counts (5,247.06±968.97 vs. 3,945.46±492.25, p<0.001). ARDS patients also exhibited significantly lower lymphocyte counts (1,030±296.25 vs. 2,304.62±367.46, p<0.001) and platelet counts (75,176.47±32,814.32 vs. 161,692.31±14,354.93, p<0.001). The NLR was markedly elevated in ARDS patients (5.35±1.24 vs. 1.74±0.29, p<0.001).

Biochemical markers such as urea (125.59±30.82 mg/dL vs. 35.23±8.06 mg/dL, p<0.001), serum creatinine (6.39±1.61 mg/dL vs. 1±0.22 mg/dL, p<0.001), and CRP (9.59±2.85 mg/dL vs. 4.31±0.75 mg/dL, p<0.001) were also significantly elevated in ARDS patients. Hypotension was more pronounced in patients with ARDS, with a considerably lower MAP of 59.71±8.56 mmHg compared to 88.85±6.08 mmHg in non-ARDS patients (p<0.001) (Table 7).

Table 7: Comparison of clinical and laboratory parameters between patients with and without ARDS

	Mean±SD		P-value
	NARDS	ARDS	
SOFA Score	3.1±0.5	17.2±5.6	<0.001
AKIN Score	13±2.26	19±3.34	<0.001
Leukocytes (cells/µL)	6533.85±3677.71	16000±3901.12	<0.001
Neutrophils (cells/µL)	3945.46±492.25	5247.06±968.97	<0.001
Lymphocytes (cells/µL)	2304.62±367.46	1030±296.25	<0.001
Platelets (PL/µL)	161692.31±14354.93	75176.47±32814.32	<0.001
Haemoglobin (mg/dl)	7.65±3.01	9.04±0.2	0.077
NLR Ratio	1.74±0.29	5.35±1.24	<0.001
Urea (mg/dl)	35.23±8.06	125.59±30.82	<0.001
Serum Creatinine (mg/dl)	1±0.22	6.39±1.61	<0.001
Serum Bilirubin (mg/dl)	0.9±0.2	6±1.86	<0.001
CRP (mg/dl)	4.31±0.75	9.59±2.85	<0.001
Hypotension (MAP)	88.85±6.08	59.71±8.56	<0.001

DISCUSSION

Our study of 50 patients with acute paraquat poisoning showed that paraquat poisoning was most prevalent in the 21-50 years, and 33 of 50 patients were below the age of 50 years. This is similar to a study from China, where the mean age of the patients was 32 years. This young age group is affected by exposure and also in terms of procurement and productivity. This study reported on the target age group by improving the management protocol and decreasing mortality.[4]

In our study, males (n=27) and females (n=23) were equally distributed. Intoxication was more common in women. But, a study by Vadlani et al. found the majority were male (n=29, 82.8%) and female (n=6, 17.1%) were the least.1 A study, that looked back at a cohort of 240 patients, found that those in the liver injury group were older, had a higher R value ([ALT/ULN]/[ALP/ULN]) (p < .001), and experienced a greater mortality rate compared to those with normal liver function.[5]

In our study, most cases occurred due to ingestion, with the most common paraquat compound abuse being paraquat dichloride 24% SL. SOFA scoring was more reliable than it correlated clearly with the duration of hospital stay, development of complications, the quantum of exposure, mechanical ventilation, pulse therapy, and haemodialysis.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Patients with ARDS had higher SOFA and AKIN scores, leucocytosis, increased neutrophil count, and increased NLR than non-ARDS patients. Hence, our study SOFA and AKIN scores predict the early development of ARDS; therefore, we can start pulse therapy as early as possible, thus reducing morbidity and mortality.

We observed a significant increase in leukocyte counts, NLR, serum creatinine, and CRP levels over time, indicating an escalating inflammatory response and renal dysfunction in these patients. Our findings align with those of Wang et al., who reported that non-survivors of paraquat poisoning had significantly higher neutrophil granulocyte ratios, leukocyte counts, and serum creatinine levels than survivors. The high predictive accuracy of NLR in their study (AUC=0.8667) supports our observation that an increased NLR correlates with a worsening prognosis.[10]

Similarly, Wilson et al. noted elevated serum creatinine levels (mean: 2.8 mg/dL) in patients with paraquat poisoning, which closely aligns with our recorded values. Their findings emphasised the role of renal dysfunction in predicting outcomes.[11] Yadla et al. emphasised the severity of AKI in paraquat poisoning cases, showing the necessity of renal replacement therapy in 95% of patients, with a mortality rate of 68%.12 Our study also identified worsening renal parameters as a significant contributor to prognosis, supporting this conclusion.

In our study, the decrease in the PaO_2/FiO_2 ratio and the drop in ABG pH showed worsening respiratory failure and metabolic acidosis. Rao et al. demonstrated that a PaO_2/FiO_2 ratio $\leq$ 197 was a strong predictor of mortality (AUC=0.924, sensitivity=97%).[13] Our findings, which show a significant decline in this ratio over 48 h, emphasise the importance of oxygenation indices in early mortality prediction.

Our study identified SOFA and AKIN scores as strong predictors of severe complications, including ARDS and the need for mechanical ventilation. The association between high SOFA scores and the need for intensive interventions aligns with previous reports. Similar to Rao et al.'s findings, a SOFA score $\geq$ 9 had high predictive accuracy (AUC=0.980) for mortality. Patients requiring mechanical ventilation had extremely poor prognoses, with all such cases resulting in death. This further confirms that early organ dysfunction scores can effectively predict outcomes and guide critical-care interventions.[13]

Our findings emphasise the role of SOFA scores in mortality prediction and suggest that patients with worsening scores should be prioritised for aggressive management. Furthermore, Wilson et al. found that low bicarbonate levels and hypokalaemia were key risk factors for mortality. Their investigations revealed an average serum creatinine of 2.8 mg/dL (+ 3.16) and bicarbonate of 17.6 mg/dL (+ 4.47).[11] Although our study did not specifically measure bicarbonate levels, the noted decrease in ABG pH indicates the presence of metabolic acidosis, which may lead to adverse outcomes.

Our study found that patients ingesting >150 mL of paraquat had the highest SOFA scores and the worst outcomes. This finding is consistent with that of Yadla et al., who reported that the amount of paraquat ingestion and latency of referral influenced survival. Their analysis showed that survival duration was significantly shorter in patients with higher paraquat exposure,[12] supporting our observation that the volume of ingestion plays a critical role in the prognosis. Zhang et al. emphasised the impact of treatment refusal, particularly hemoperfusion, on mortality outcomes.[14] Although our study did not directly assess treatment refusal, the need for intensive interventions, such as haemodialysis, was significantly higher in patients with worsening SOFA and AKIN scores, reinforcing the importance of early and aggressive management.

CONCLUSION

PQ poisoning is associated with leucocytosis, neutrophilia, and lymphopenia. NLR, leukocyte count, and neutrophil count are excellent prognostic markers for acute PQ poisoning. Our findings show that SOFA and AKIN scores at 48 h were significantly associated with the need for mechanical ventilation, haemodialysis, pulse therapy, and the development of ARDS following PQ poisoning. Given the absence of a specific antidote, increased clinician awareness and the availability of laboratory diagnostic methods are vital for improving the management and outcomes of patients with PQ poisoning.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556

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ISSN (O): 2687-5365; ISSN (P): 2753-6556