Section: Emergency Medicine

Original Research Article

THE CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH PARAQUAT POISONING: A PROSPECTIVE STUDY IN SOUTHERN INDIA

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ABSTRACT

Background: Paraquat is a highly toxic herbicide responsible for severe poisoning and death, particularly in agricultural regions of India. Its easy availability and limited regulation make accidental and intentional ingestion a serious public health issue. This study aimed to describe the clinical profile, organ involvement, and outcomes of paraquat poisoning in Southern India. **Materials and Methods:** A prospective observational study was conducted at a tertiary care hospital, enrolling 37 patients with confirmed paraquat ingestion. Data on demographics, exposure, symptoms, organ dysfunction, investigations, management, and outcomes were collected and analysed using descriptive statistics. **Result:** Of the 37 patients, 51.4% were aged 51–80 years, 35.1% were >80 years, and 67.6% were male. Most (81.1%) presented after >2 hours of ingestion, and 54.1% consumed >50 mL. Direct ingestion occurred in 40.5%, while 48.6% ingested with alcohol. Common symptoms were vomiting (81%), nausea (48.6%), and abdominal pain (35%). Organ dysfunction frequently involved kidneys (81%) and gastrointestinal tract (78.4%), followed by lungs (48.6%), liver (40.5%), and cardiovascular system (40.5%). Abnormal renal function tests occurred in 73%, arterial blood gas in 51.4%, and liver function in 48.6%. Haemodialysis was required in 10.8%. Mortality was highest in patients with >3 organ failures (76.9%) and lung involvement (72.2%). Overall, 19 patients (51.4%) died, 8 (21.6%) left against medical advice, and 10 (27%) survived. **Conclusion:** Paraquat poisoning carries very high mortality, especially with direct ingestion, lung or kidney involvement, and multiorgan dysfunction. Early medical care and preventive strategies are essential to improve survival.

INTRODUCTION

Paraquat (1,1′-dimethyl-4,4′-bipyridinium dichloride) is a widely used non-selective contact herbicide known for its rapid weed control and low cost.[1] It is approved for use in over 100 countries and is frequently used in India, particularly in the agricultural areas of Southern India.[2] Despite its usefulness in farming, paraquat is extremely toxic to humans and animals.[3] Ingestion of as little as 10–15 mL of a 20% commercial preparation, equivalent to about 3–5 mg/kg, can be fatal, with case fatality rates ranging from 50% to over 90%.[4] In India, most deaths are due to deliberate ingestion in acts of self-harm, aided by its easy availability and low price.[5] Accidental poisonings also occur, usually from unsafe storage or mistaken ingestion.[6] Some countries require the addition of a blue dye, a strong odour, and an emetic to prevent accidental ingestion, but these measures are less effective in deliberate cases.[7] In India, regulatory measures remain minimal, allowing continued widespread access.[8]

The toxic effects of paraquat are caused by intracellular redox cycling, which produces large amounts of reactive oxygen species (ROS) and depletes the antioxidant NADPH.[9] This results in oxidative damage, lipid peroxidation, mitochondrial injury and cell death. The lungs are most affected because alveolar cells actively take up and concentrate paraquat, leading to acute inflammation and later irreversible pulmonary fibrosis, the main cause of death in survivors of the initial phase.[10] Other organs are also damaged, including the gastrointestinal tract, kidneys, and liver. The severity and combination of organ injuries significantly influence outcomes.

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There is no specific antidote for paraquat poisoning; therefore, treatment is supportive. If patients present early, gastrointestinal decontamination with activated charcoal or fuller's earth can reduce absorption. Hemoperfusion or haemodialysis may remove paraquat from the blood if started within the first few hours, though this is often not possible in practice.[4,6] Supportive measures include fluid therapy, nutritional support, and careful oxygen use, as high oxygen levels can worsen lung injury.[9] Immunosuppressants and antioxidants have been tried, but their benefit remains unproven.[5,8]

The prognosis depends on the dose ingested, the time before treatment, and the development of organ failure. Acute kidney injury or respiratory failure are strong predictors of poor outcomes.[3,10] Prognostic tools such as the Severity Index of Paraquat Poisoning (SIPP) and Sequential Organ Failure Assessment (SOFA) score can help estimate risk.[7,9] Despite its high burden, prospective regional studies in India are limited. This study aimed to describe the sociodemographic profile, clinical features, organ involvement, and outcomes of acute paraquat poisoning in Southern India and to identify factors linked to mortality to improve local management strategies.

MATERIALS AND METHODS

This prospective observational study was conducted in the emergency department and intensive care unit of a tertiary care teaching hospital in Southern India, which caters to a largely rural population from agricultural districts, such as Thanjavur, Thiruvarur, Nagapattinam and Cuddalore. The study population included 37 consecutive patients with a confirmed history of paraquat ingestion. Ethical approval was obtained from the Institutional Ethics Committee, and written informed consent was obtained from each participant or their legally authorised representative before enrolment.

Inclusion and exclusion Criteria

All patients aged ≥ 18 years with a confirmed history of paraquat ingestion were included in the study, with confirmation based on the patient's statement, information from accompanying relatives, or presentation of the container containing the paraquat formulation. Patients were excluded if they had a known history of co-ingestion of other significant poisons or if the diagnosis of paraquat poisoning could not be established.

Methods: The study collected information on patient demographics, exposure details, clinical features, organ involvement, investigation results, management, and outcomes. Data were obtained prospectively from the time of admission until discharge or death using a standardised proforma, supplemented by a review of case records, clinical notes, and investigation reports. The demographic details included age, gender, occupation, and place of residence. Exposure details included the estimated amount of paraquat consumed, mode of ingestion, and time to hospital admission. Clinical presentation was documented using a checklist of symptoms, and organ dysfunction was assessed for six major systems based on clinical and laboratory findings on the third day after ingestion.

Laboratory and imaging results were recorded as normal or abnormal according to the institutional reference ranges. Management details, including whether haemodialysis was performed, were noted. The primary outcome was classified as alive and stable, discharged against medical advice, or death due to cardiac or respiratory arrest, as determined by the patient's status on day seven or earlier if a terminal event occurred.

Statistical analysis: The collected data were analysed using IBM SPSS Statistics v27. Descriptive statistics were used to summarise categorical variables, which were presented as frequencies and percentages.

RESULTS

Among the 37 patients, most were aged 51–80 years (51.4%), followed by >80 years (35.1%) and 20–50 years (13.5%), with males comprising 67.6% of the total. The majority presented after more than two hours (81.1%), with 54.1% consuming >50 mL of paraquat; 48.6% ingested it mixed with alcohol, 40.5% directly, and 5.4% mixed with other substances or unspecified. Vomiting (81%), nausea (48.6%), and abdominal pain (35%) were the most frequent symptoms, and kidney (81%) and gastrointestinal tract (78.4%) involvement were the most common. Abnormal investigations included renal function tests (73%), arterial blood gas analysis (51.4%), and liver function tests (48.6% of patients). Haemodialysis was required in 10.8% of the patients [Table 1].

Table 1: Distribution of demographic, exposure, clinical, and organ involvement characteristics

Variables		N (%)
Age group (years)	20 to 50	5 (13.5%)
	51 o 80	19 (51.4%)
	>80	13 (35.1%)
Gender	Male	25 (67.6%)
	Female	12 (32.4%)
Duration (hours)	<2	7 (18.9%)
	>2	30 (81.1%)
Location	Others	19(51.4%)
	Thanjavur	18(48.6%)

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Amount of poison (ml)	<50	13 (35.1%)
	>50	20 (54.1%)
	Unknown Quantity	4 (10.8%)
Mode of consumption	Mixed with alcohol	18 (48.6%)
	Mixed with others	2 (5.4%)
	Direct	15 (40.5%)
	Not available	2 (5.4%)
Symptoms	Breathlessness	5 (13.5%)
	Oral pain	12 (32.4%)
	Chest pain	6 (16.2%)
	Abdominal pain	13 (35%)
	Jaundice	2 (5.4%)
	Decreased urine output	2 (5.4%)
	Skin or mucus membrane burns	0
	Nausea	18 (48.6%)
	Vomiting	30 (81%)
Name of the organs	Lungs	18 (48.6%)
	CVS	15 (40.5%)
	Kidney	30 (81%)
	GI tract	29 (78.4%)
	Liver	15 (40.5%)
	Skin	1 (2.7%)
Name of investigation	ABG	19 (51.4%)
	CBC	6 (16.2%)
	RFT	27 (73%)
	LFT	18 (48.6%)
	CXR	12 (32.4%)
	USG	11 (29.7%)
Haemodialysis	Yes	4 (10.8%)
	No	33 (89.2%)
Outcome	Alive and stable	10(27%)
	AMA (against medical devices)	8(21.6%)
	Arrest	19(51.4)

Among patients presenting within two hours of ingestion, 85.7% had fewer than three organ dysfunctions, 14.3% showed no dysfunction, and none developed more than three dysfunctions. In contrast, among those presenting after two hours, 53.3% had fewer than three dysfunctions, 43.3% developed more than three dysfunctions, and 3.3% had no dysfunction (p=0.071).

Patients aged 51–80 years had the highest mortality rate, with 57.9% of arrests, followed by those >80 years (53.8%), whereas most in the 20–50 years' group left against medical advice (60%) (p=0.203). Female patients had a higher arrest rate (75%) than male patients (40%) (p=0.129). Presentation time showed little influence on outcome, with 53.3% of those presenting after two hours and 42.9% of those within two hours succumbing (p=0.851). Direct ingestion was associated with the highest mortality (73.3%), while ingestion with alcohol showed 38.9% deaths, and mixed with other substances 50% (p=0.093). Patients with more than three organ dysfunctions had markedly poorer outcomes, with a mortality rate of 76.9% compared to 40.9% in those with fewer dysfunctions (p=0.011). Kidney involvement was associated with a 60% mortality rate (p=0.067). Liver dysfunction showed a similar distribution of deaths (53.3%) compared to those without (50%) (p=0.975). Lung involvement strongly correlated with mortality, with 72.2% deaths versus 31.6% in those without (p=0.01). Cardiovascular dysfunction also showed higher mortality (66.7%) than that in patients without (40.9%) (p=0.299) [Table 2].

Table 2: Association of demographic, clinical, and organ dysfunction variables with patient outcomes in paraquat poisoning

Variables		**Outcome**			**p-value**
		Alive and stable	**AMA**	**Arrest**	
Age group (years)	20 to 50	1(20%)	3(60%)	1(20%)	0.203
	51 o 80	6(31.6%)	2(10.5%)	11(57.9%)	
	>80	3(23.1%)	3(23.1%)	7(53.8%)	
Gender	Male	8(32%)	7(28%)	10(40%)	0.129
	Female	2(16.7%)	1(8.3%)	9(75%)	
Duration (Outcome of the 7th day)	<2 hours	2(28.6%)	2(28.6%)	3(42.9%)	0.851
	<2 hours	8(26.7%)	6(20%)	16(53.3%)	
Mixed With Alcohol		5(27.8%)	6(33.3%)	7(38.9%)	0.093
Mixed With Others		1(50%)	0	1(50%)	
Direct		2(13.3%)	2(13.3%)	11(73.3%)	
NA		2(100%)	0	0	
Organ dysfunction	<3	9(40.9%)	4(18.2%)	9(40.9%)	0.011
	>3	1(7.7%)	2(15.4%)	10(76.9%)	
	No	0	2(100%)	0	

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Kidney	Yes	6(20%)	6(20%)	18(60%)	0.067
	No	4(57.1%)	2(28.6%)	1(14.3%)	
Liver	Yes	4(26.7%)	3(20%)	8(53.3%)	0.975
	No	6(27.3%)	5(22.7%)	11(50%)	
Lungs	Yes	1(5.6%)	4(22.2%)	13(72.2%)	0.01
	No	9(47.4%)	4(21.1%)	6(31.6%)	
CVS	Yes	3(20%)	2(13.3%)	10(66.7%)	0.299
	No	7(31.8%)	6(27.3%)	9(40.9%)	

DISCUSSION

This study aimed to analyse the clinical profile, organ dysfunction patterns, and outcomes of patients with pesticide poisoning. The majority of patients were aged 51–80 years (51.4%), followed by those over 80 years (35.1%) and 20–50 years (13.5%), with males comprising 67.6% of the cases. Similarly, Chen et al. found that the mean age of exposure was 56.4 ± 16.8 years, with males comprising 68.9% of cases. Paraquat exposure was associated with the highest mortality rate (77.1%) among all agents.[11] Rahman et al. found that among paraquat poisoning cases, 66.7% were male, and most were older than 30 years.[12] Older age and male predominance were consistent across studies, with paraquat exposure being associated with the highest mortality burden.

Our study showed that most patients presented after two hours of ingestion, but timing had little effect on the outcome. Mortality was highest with direct ingestion (73.3%) than with alcohol-mixed (38.9%) and other mixtures (50%). Similarly, Tan et al. reported that most exposures were intentional (69.6%), with 72.2% of patients presenting to the hospital within six hours of ingestion.[13] Goyal et al. found that survivors reached the hospital much earlier, with a mean duration of 17.26 ± 17.23 hours (median 14 hours), compared to non-survivors who presented after a longer interval, with a mean of 80.18 ± 90.07 hours (median 48 hours).[14] In contrast, Dhanarisi et al. reported in a meta-analysis that alcohol co-ingestion in pesticide self-poisoning was associated with an increased risk of death [odds ratio (OR) 4.9, 95% confidence interval (CI) 2.9-8.2, p<0.0001].[15] Timing influenced survival in some studies, but in our study, direct ingestion carried the highest mortality risk.

In our study, the main symptoms were vomiting, nausea, and abdominal pain, with kidney dysfunction being the most common, followed by gastrointestinal, lung, liver, and cardiovascular involvement. Mortality was higher (76.9%) in patients with > 3 organ dysfunctions than in those with fewer organ dysfunctions (40.9%). Similarly, Khan et al. found that the most frequent symptom was vomiting with abdominal pain in 66.7% of patients, followed by throat pain in 30% of patients. The leading clinical sign was oral mucosal excoriation in 73.3% of cases, followed by jaundice in 36.7%.[16] Reddy et al. found that in a case series of 15 patients, renal involvement was reported in 93.3%, followed by lung and liver involvement in 60% each.[17] Shi et al. found in a cohort study that 70.3% of non-survivors had dysfunction in more than three organs, whereas only 38.7% of survivors showed similar involvement (p<0.01).[18] Gastrointestinal and renal manifestations were dominant, with multi-organ dysfunction strongly linked to adverse prognosis across multiple studies.

Our study showed abnormal findings in renal function tests (73%), arterial blood gas analysis (51.4%), and liver function tests (48.6%). Chest radiography (32.4%) and ultrasound (29.7%) also revealed abnormalities. Haemodialysis was required in 10.8% of patients. Similarly, Ravichandran et al. found that acute kidney injury occurred in 81.8% of patients, with 56.3% requiring dialysis, while liver abnormalities (33.3–46.6%) and lung injury (61.8%) with chest X-ray infiltrates were also common.[19] Laboratory and imaging abnormalities were frequent, and dialysis was occasionally required, with comparable organ involvement reported in other studies.

In our study, the outcomes showed that 27% survived, 21.6% left against medical advice, and 51.4% died. Mortality was particularly high in patients with lung involvement (72.2%) and cardiovascular dysfunction (66.7%) compared with that in patients without these conditions. Similarly, Halesha et al. found that 72% of patients died, 18% achieved complete recovery, and 10% left against medical advice.[20] Ravichandran et al. found that lung injury was strongly associated with mortality, with 88.2% of affected patients dying compared to 47.6% without lung injury (p=0.001).[19] Mortality was high overall, with lung and cardiovascular dysfunction serving as strong predictors of poor clinical outcomes.

Our study highlights that pesticide poisoning carries high mortality rates, particularly with paraquat and multi-organ failure. Late arrival, direct intake, and lung or heart involvement worsen outcomes, highlighting the importance of early care and prevention.

Limitations: This study included a small number of patients from a single hospital; therefore, the findings may not apply to all settings. Blood paraquat levels, which are important for predicting outcomes, could not be tested due to limited resources. In addition, long-term effects and late complications were not studied, as follow-up was performed only during hospital stay.

CONCLUSION

Paraquat poisoning causes severe illness with high mortality rates, particularly among patients who consume larger amounts and those with lung and

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kidney damage. Most patients presented to the hospital late after ingestion, which reduced the chances of effective treatment. Symptoms such as vomiting, nausea, and abdominal pain were common at presentation, whereas multiorgan dysfunction was strongly associated with poor outcomes. Patients with more than three organ failures had a much higher risk of death than those with fewer complications. Lung involvement, in particular, showed a clear association with mortality, highlighting the importance of early identification and management of respiratory disorders. Although supportive care and dialysis were used in some patients, the overall survival rate remained low. These results emphasise the urgent need for preventive strategies, strict regulation of paraquat availability, and early medical attention to improve survival in affected individuals.

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