ORIGINAL ARTICLE

A Study of Paraquat Poisoning Presentation, Severity, Management and Outcome in a Tertiary Care Hospital: Is There a Silver Lining in the Dark Clouds?

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Abstract

Introduction: Accidental or intentional ingestion of paraquat leads to many local and systemic effects and the mortality rate is very high. There is limited data from North India and our objectives were to study the spectrum of presentation, treatment given, and its relation with outcome in a tertiary care setting.

Materials and methods: This retrospective observational study was conducted after ethical approval and data regarding demography, clinical features, duration of presentation, organ involvement, renal replacement therapy (RRT), management, and outcome was collected. Statistical analysis was done by calculating mean and standard deviation (SD). Chi-square (χ^2) test was applied to categorical variables and the Fisher exact test was used when the expected frequency was less than 5.

Results: The study population consisted of 91 male (84%) and 18 female patients. Out of 109 patients, 13 survived (12%) and 88% had a fatal outcome. Nearly 92% of patients belonged to rural background, and 68% were of younger (<30 years) age group. Age, gender, occupation, and amount taken did not have any significant relation with mortality. Patients having metabolic acidosis (58.7%), altered renal (75.2%), and hepatic function (62.3%) at presentation had a statistically significant relation with mortality. Duration of presentation was significantly lesser in patients who survived (17.26 ± 17.23, median 14 hours vs 80.18 ± 90.07, median 48 hours) compared to patients who did not survive. Renal replacement therapy ($n = 57$) had no relation with mortality whereas 36% of the patients who received hemoperfusion (HP) survived ($p = 0.03$).

Conclusion: Treatment should be started early as the duration of the presentation has a significant association with the outcome. Currently there is no antidote available. Supportive treatment includes oxygenation, immunosuppression, antioxidants, RRT, and HP wherever the resources are available.

Keywords: Hemoperfusion, Immunosuppression, Mortality, Paraquat, Poisoning, Renal failure.

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Highlights

Duration of presentation from the time of poisoning to initiation of treatment has a significant relationship with mortality. Patients presenting with metabolic acidosis, renal failure, and respiratory distress warranting invasive or non-invasive ventilation (NIV) had poor prognosis. Hemoperfusion (HP) when started early within four hours may improve the outcome. Patients were of young age, and most were students or of agriculture occupation, so their mental health and stressors should be looked into so that remedial actions can be taken at an earlier time. Alternative less toxic herbicides and strict government regulations may hold the key to decreasing the accidental or intentional intake of paraquat poisoning.

Introduction

Paraquat (1,1′-dimethyl-4, 4′-bipyridinium) is a commonly used herbicide, and accidental or intentional intake of this herbicide leads to ulceration of skin around the mouth, oral cavity, pharynx, and esophagus. Ingestion of paraquat also leads to respiratory failure, cardiac involvement, renal failure, hepatic derangements, and pulmonary fibrosis (long term).[1] Paraquat has been classified variably as class II or moderately hazardous chemical by the World Health Organization while the Pesticide Action Network (PAN) International has categorized it as a highly hazardous pesticide.[2,3] Paraquat is banned in more than 60 countries, including the United Kingdom, China, Brazil, and members of the European Union, Malaysia, and Sri Lanka.[4] Paraquat metabolism leads to the generation of free radicals, which in turn damage the cellular organelles and cellular functions, thus causing irreparable loss of structure and function of many vital systems. Paraquat is structurally similar to polyamines, hence cells of pulmonary alveoli selectively accumulate polyamines as well as paraquat resulting in delayed pulmonary dysfunction ultimately causing pulmonary fibrosis.[5] There is a lack of effective antidote and specific treatment for paraquat poisoning. The mortality rate is high and currently, there are no recommendations regarding treatment of this poison. Some

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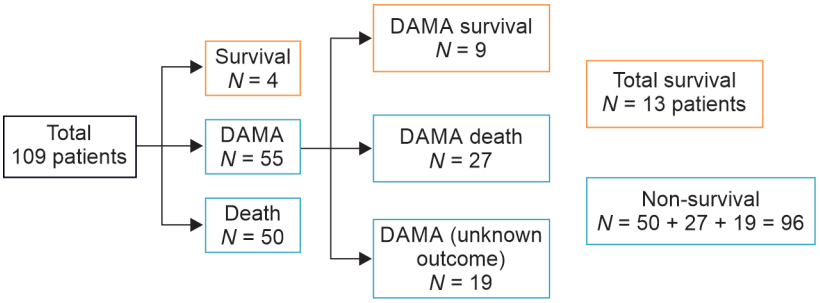

Fig. 1: Distribution of patient population with respect to survival

studies have reported that consumption of paraquat >40 mg/kg (>10 mL, 20% solution) results in multi-organ failure and ultimately mortality within a few days of presentation, while intake of <20 mg/kg (<5 mL) may cause mild symptoms and the patient may recover.[6,7] Paraquat dose of 20–40 mg/kg (5–10 mL) may cause local and systemic involvement followed by multiorgan dysfunction syndrome and ultimately may result in high morbidity. The patient may develop delayed pulmonary fibrosis within 2–4 weeks.[1] The treatment mostly includes gastric lavage with adsorbents such as activated charcoal, systemic steroids, immune suppressants (cyclophosphamide), or elimination of paraquat through specific adsorbent filters (e.g., HA 320).[8] Though there is some available evidence that has shown the benefit of early HP (with toxin-specific cartridges) within four hours of consumption yet there is a lack of conclusive evidence.[9] Although paraquat poisoning is quite common in India, there are few studies from North India describing the presentation and outcome.[5,6] Hence, this study was conducted to describe the clinical presentation, signs and symptoms, management, and outcome following paraquat poisoning at our tertiary care hospital. Various treatment modalities used and their relation with outcome were also described.

Materials and Methods

This retrospective observational study was conducted at our tertiary care institute after ethical approval (IEC No. 2023-828) from the Hospital ethical committee. All patients diagnosed with paraquat ingestion during the study period of two years (2022–2023) were included in the study. A structured proforma was developed and used to collect data regarding the demographics, signs and symptoms, complications, outcomes, management, laboratory, and radiological findings from hospital records. Patients were diagnosed as having hepatic and renal failure based on elevated laboratory values as well as clinical characteristics and diagnoses mentioned in records. As per our hospital laboratory standards, serum creatinine > 1.2 mg/dL, blood urea > 50 mg/dL, total bilirubin > 1.2 mg/dL, direct bilirubin > 0.3 mg/dL, SGOT > 40U/L, SGPT > 41 U/L were considered out of range. The amount of paraquat ingested was also noted, wherever the information was available. Arterial blood gases at admission as well as data regarding organ failure, oxygen support, renal replacement therapy (RRT), inotropic support during management, and its relation with outcome was also collected. Additional data of those patients who received HP (HA 320 cartridge) was collected and the outcome was noted.

Data was described in terms of range, mean ± standard deviation (±SD), median and interquartile range (IQR), frequencies (number of cases), and relative frequencies (percentages) as appropriate. To determine whether the data is normally distributed, a Kolmogorov-Smirnov test was used. Comparison of quantitative variables between the study groups was made using the student *t*-test and Mann–Whitney test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi-square (χ^2) test was performed and the Fisher exact test was used when the expected frequency was less than 5. A probability value (*p*-value) less than 0.05 was considered statistically significant. All statistical calculations were done using Statistical Package for the Social Science (SPSS 20.0) version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

Results

The patient population consisted of 109 patients including 91 male (84%) and 18 female patients. Most of the patients (n = 100, 91.7%) belonged to rural backgrounds. Patients who were discharged against medical device were contacted telephonically and it was observed that thirteen patients survived (12%) and 96 patients (88%) had fatal outcomes (Fig. 1). The demographic distribution of patients was variable, largely belonging to districts Hoshiarpur (11), Ludhiana (10), Sangrur (8), Jalandhar (8), Firozpur (6), Muktsar (6), as shown in Figure 2. Eleven patients belonged to neighboring states of Punjab and the native place of 8 patients was unknown.

Table 1 shows the distribution of patients with respect to age. Most of the patients belonged to the younger age group with nearly 68% of patients of <30 years followed by 24% patients of 31–40 years of age (Table 1), the youngest patient being 15 years old and the eldest being 67 years old. The Mean age of patients who survived was 25.69 ± 6.46 (Median = 23.00, IQR: 20–34) as compared to 28.23 ± 10.07 (Median = 26.00, IQR: 21–32) in patients who did not survive. Most of the patients belonged to agriculture-related occupations (32%) followed by the dependent (students) category (31.2%) as shown in Figure 3. Age, gender, place of residence, and occupation of patients did not have any significant relation with mortality in our study. The probable amount of paraquat ingestion was known only in 21 patients as shown in Table 2. The amount of paraquat ingestion did not have a significant relationship with mortality. Data was also collected to note the time interval from the time of ingestion of paraquat to presentation to the hospital and it had significant relation with mortality ($Z = 3.571$, p-value = 0.001). Time interval in hours was significantly lower, i.e., (17.26 ± 17.23; Median = 14.00, IQR = 2.7375–23.75) in survivors than in non-survivors (80.18 ± 90.07; Median = 48.00, IQR = 23–108.2). The shortest time interval recorded was 0.40 hours (24 minutes, the patient survived) and the longest was 456 hours (19 days, the patient did not survive). Figure 4 shows that a maximum number of patients (n = 60) arrived >24 hours later from the time of ingestion of paraquat and mortality was highest in this group.

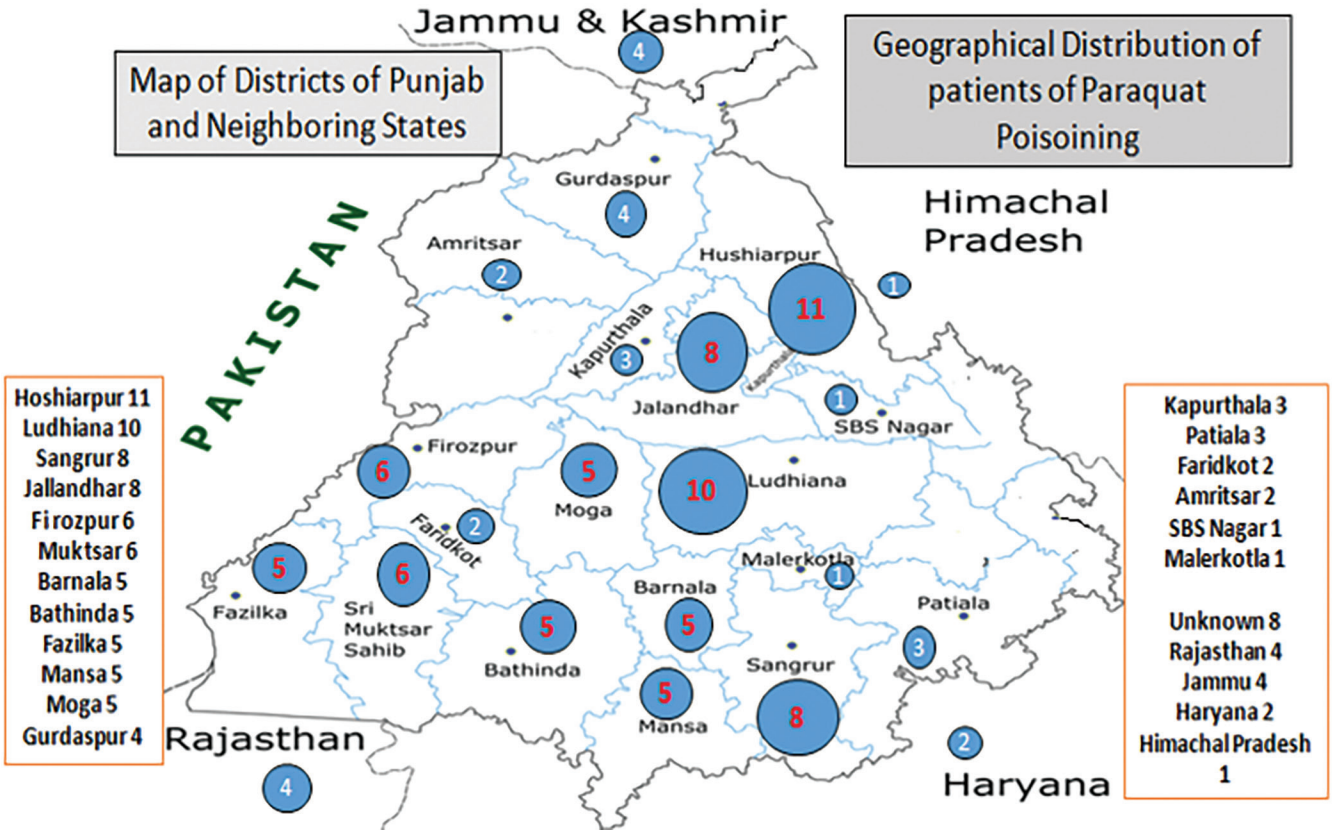

Fig. 2: Geographic distribution of patient population

Table 1: Distribution of patients with respect to age

Age in years	*N*	*Outcome*		*Chi-square value*	*p-value*
		Survivors	*Non-survivors*		
≤20	23 (21.1%)	4 (30.8%)	19 (19.8%)	2.387	0.665
21–30	51 (46.8%)	5 (38.5%)	46 (47.9%)		
31–40	26 (23.9%)	4 (30.8%)	22 (22.9%)		
41–50	6 (5.5%)	0	6 (6.3%)		
>50	3 (2.8%)	0	3 (3.1%)		
Total	109	13	96		

Common presenting symptoms were nausea and vomiting (61.4%), oral ulceration (42.2%), breathing difficulty (36.7%), difficulty in swallowing (30%), hypotension (30%), decreased urine output (26%) and gastrointestinal bleeding as shown in Figure 3. Vital parameters and laboratory investigations at presentation were analyzed for any relation with mortality and it was observed that mean systolic blood pressure (Mean = 126.69 ± 22.11 mm Hg), diastolic blood pressure (Mean = 77.37 ± 11.86 mm Hg), and heart rate (Mean = 96.11 ± 21.94) of the patients did not have significant relation with mortality. Parameters such as respiratory rate, pH, pCO_2, HCO_3, lactate, blood urea, serum creatinine, total bilirubin, direct bilirubin, SGOT, SGPT, alkaline phosphatase, and total leucocyte count had significant relation with mortality and were significantly altered in non-survivors as shown in Table 3. Patients having metabolic acidosis ($n = 64$), altered renal ($n = 82$) and hepatic function ($n = 68$), vasopressor support, intubation, and mechanical ventilation at presentation also had a statistically significant relation with mortality (Table 4).

Out of 109 patients, 57 (52.3%) patients received RRT in the form of hemodialysis (HD)/sustained low-efficiency dialysis (SLED)/ continuous renal replacement therapy (CRRT), and as shown in Figures 5 and 6. Out of these 57 patients who received RRT, 7 patients survived and the relation of RRT to mortality outcome was found to be non-significant. Most of the patients presented late (more than 4 hours after paraquat intake) and only 12 patients (11%) presented within the time window and were eligible to receive HP with an HA 230 cartridge, out of which 4 patients (33.3%) survived and the relation of HP with survival outcome was found to be significant ($\chi^2 = 5.8827$, $p = 0.015$).

It was observed in our study that when divided into two groups; patients who received HP and patients who did not receive HP, patients were comparable in all characteristics except that patients in the latter (no HP) group had a higher number of patients with renal failure ($p = 0.032$), hepatic failure ($p = 0.009$), metabolic acidosis and elevated serum lactate indicating higher severity and longer duration of presentation.

Among adjuvant treatments, 95 (87%) patients received steroids, 62 (56.8%) cyclophosphamide, 94 (86.2%) NAC and 87 (79.8%) patients received Vitamin C treatment along with supportive treatment. These modalities of treatment had no significant relation with mortality.

Out of the total patient population, 13 patients survived and among survivors, 9 patients were of the younger age group

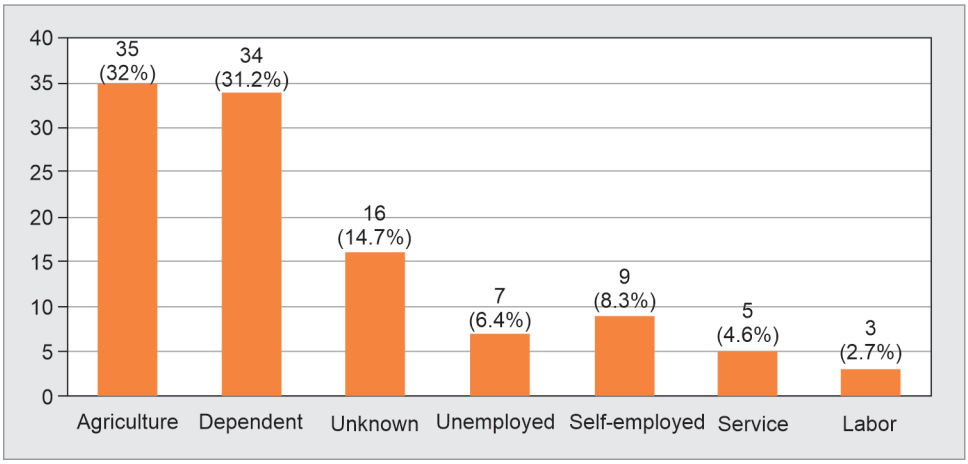

Fig. 3: Distribution of patients with respect to occupation

Table 2: Relationship between estimated amount of paraquat ingestion and outcome

Amount of paraquat	*Survivor N*	*Non-survivors N*	*Total (%)*	*Chi-square value*	*p-value*
<5 mL	2	6	8 (38.1%)	1.219	0.748
5–10 mL	0	4	4 (19.0%)		
10–20 mL	1	3	4 (19.0%)		
>20 mL	1	4	5 (23.8%)		
Total	4	17	21		

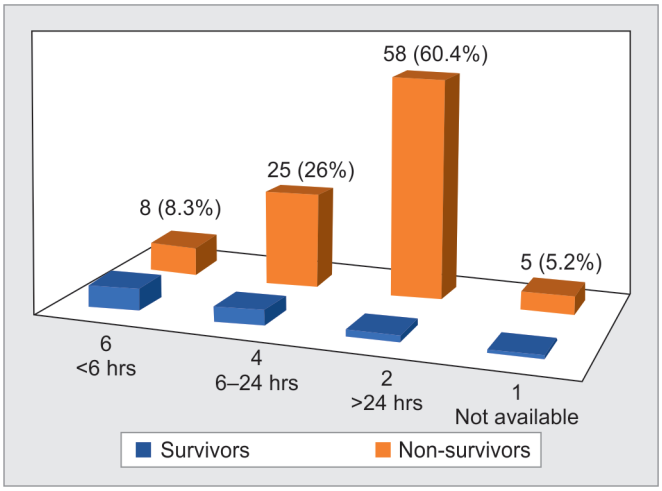

Fig. 4: Relation between time lag to presentation to hospital and outcome

(<30 years), 85% ($n = 11$) were male, 50% ($n = 6$) were dependent/student and all of them belonged to rural backgrounds. Four patients presented within 6 hours and another 4 patients presented between 6 and 24 hours. The patients who survived did not have shock and respiratory failure at presentation. None of them needed NIV, non-rebreathing masks (NRBM), or intubation. Among the survival group, 3 patients had metabolic acidosis and deranged liver function tests (LFTs), whereas 7 patients had deranged renal function tests (RFTs) who eventually received RRT and recovered. Among survivors, 4 patients had received HP. Cyclophosphamide and steroids were the adjuvant therapies received by these patients.

Despite having a smaller number of survivals in paraquat poisoning patients in our study, an attempt was made to apply forward stepwise logistic regression analysis to the data taking into consideration the significant parameters such as duration of presentation, blood urea, serum creatinine, lactate, bicarbonate, direct bilirubin, vasopressors and HP as shown in Table 5. On forward stepwise logistic regression analysis, duration of presentation and lactate were the variables that showed a strong association with survival.

Discussion

Paraquat is widely manufactured as paraquat dichloride and paraquat bismethylsulfate and it is a synthetic, non-selective herbicide with no known antidote. Paraquat dichloride (24% strength) is a commonly used herbicide in India and its accidental and intentional ingestion has a high case fatality rate (60–80%).[10] Its ingestion causes erosion of the gastrointestinal tract and ulcerations on the mouth and the tongue also called paraquat tongue. Oral and esophageal ulceration may lead to esophageal perforation, mediastinitis, and pneumomediastinum. After ingestion, nearly 20% of paraquat is absorbed by the stomach and is concentrated 10–20 times in pulmonary alveoli leading to the production of toxic reactive oxygen species causing immediate and long-term pulmonary damage (alveolitis and fibrosis). Plasma levels peak at 5–6 hours and then rapidly decrease due to redistribution to tissues. Paraquat causes renal injury as it is actively eliminated by the kidney causing free oxygen radical injury to proximal tubular epithelial cells whereas hepatocellular injury occurs due to mitochondrial damage and endoplasmic reticulum degranulation.[1,7,10]

Mortality rate in our study was slightly higher (88%) as compared to other studies, which may be due to the exclusion of discharge against medical advice patients in earlier studies and delayed presentation.[9,11] Our patient population consisted mostly of younger age group (68% in <30 years), male gender (84%), and most of them belonged to rural background (92%). Occupation was variable with most involved in agriculture (32%) or were

Table 3: Laboratory parameters of patients at presentation and its relation with outcome

	Survivors				*Non-survivors*					
	Mean	*SD*	*Median*	*IQR*	*Mean*	*SD*	*Median*	*IQR*	*Z*	*p-value*
RR (/min)	21.38	1.71	20.00	20–23	24.20	8.17	24.00	22–24	2.619	0.009
pH	7.42	0.03	7.41	7.39–7.46	7.36	0.12	7.39	7.33–7.44	1.732	0.083
pCO_2 (mm Hg)	36.36	4.54	37.00	34–39	32.36	8.73	32.00	28–37	2.033	0.042
pO_2 (mm Hg)	92.64	17.07	91.00	78–107	100.21	50.14	92.00	70–111	0.016	0.988
HCO_3 (mmol/L)	23.70	2.94	23.60	21.5–24.8	21.17	22.53	19.40	14.9–23.3	2.616	0.009
Lactate (mmol/L)	1.08	0.39	0.90	0.8–1.3	3.92	3.90	1.95	1.3–5.63	3.754	0.001
Blood urea (mg/dL)	54.36	52.04	36.00	24–58	99.82	72.82	74.00	47.5–147.5	2.490	0.013
Serum creatinine (mg/dL)	2.21	2.34	0.95	0.83–4.39	5.04	3.70	4.26	2.08–7.26	3.129	0.002
Total bilirubin (mg/dL)	1.60	1.87	0.84	0.36–2.31	3.83	4.09	2.98	0.68–5.13	1.890	0.059
Direct bilirubin (mg/dL)	0.80	1.50	0.22	0.12–0.75	4.66	18.16	1.85	0.22–4.26	2.247	0.025
SGOT (U/L)	46.10	52.42	30.50	26.5–38	176.10	238.02	105.00	41.5–230.5	3.459	0.001
SGPT (U/L)	62.80	79.60	37.50	26–61.5	187.13	196.58	125.00	40.5–281.75	2.700	0.007
Alkaline phosphatase (IU/L)	100.40	71.73	74.50	63.25–107	173.93	136.49	124.00	77–242	2.153	0.031
TLC ($\times 10^9$/L)	9.78	2.62	9.10	7.8–10.77	16.85	12.60	14.54	10.15–19.4	2.906	0.004

Table 4: Relation between organ failure and support requirement and mortality outcome

		Outcome			
Organ failure and support requirement	*N*	*Survivors*	*Non-survivors*	*Chi-square value*	*p-value*
Metabolic acidosis	64 (58.7%)	3 (23.1%)	61 (63.5%)	7.734	0.007
Renal failure	82 (75.2%)	3 (23.1%)	79 (82.3%)	21.544	0.001
Hepatic failure	68 (62.4%)	3 (23.1%)	65 (67.7%)	9.719	0.004
Vasopressors	37 (33.9%)	0	37 (38.5%)	7.585	0.004
Intubation and mechanical ventilation	38 (34.9%)	0	38 (39.6%)	7.9	0.004

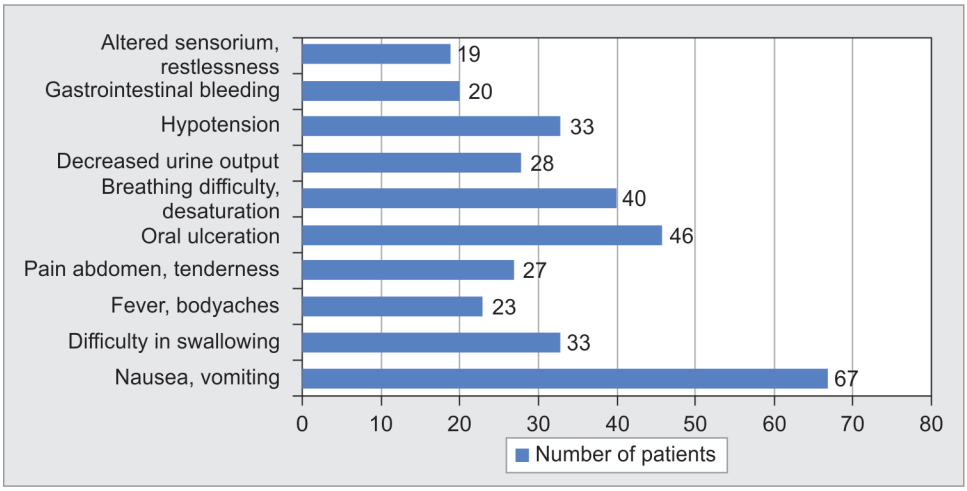

Fig. 5: Distribution of clinical features and presenting symptoms

students (31.2%). Age, gender, place of residence, and occupation of patients did not have any significant relation with mortality in our study. Derangement of parameters such as respiratory rate, pH, pCO_2, HCO_3, lactate, RFTs and LFTs at presentation had a significant relation with mortality. Similar to other studies higher mortality in our study is also significantly associated with delayed presentation.[5,11] Maximum number of patients (60) arrived >24 hours later than the time of ingestion of paraquat and mortality was highest in this group.

There was limited data about the quantity of paraquat ingestion and it did not have a significant relationship with the outcome which may be due to the unavailability of proper estimation of the amount ingested as well as incomplete data. Diagnosis is based on careful history taking along with the availability of causal agents

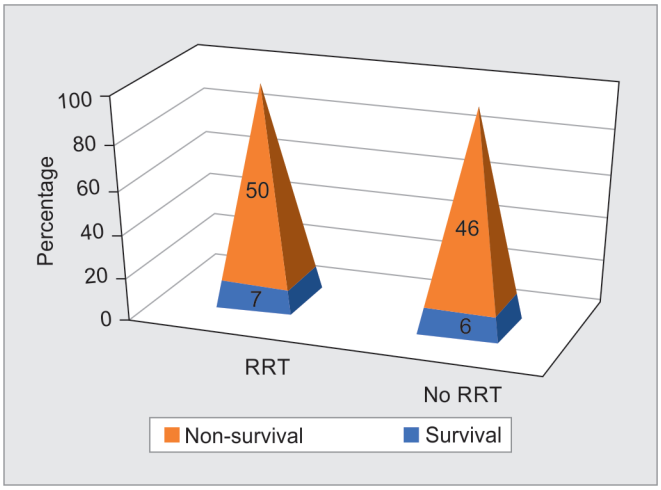

Fig. 6: Relationship between renal replacement therapy and outcome

Table 5: Forward stepwise logistic regression analysis

			95% CI for odd ratio	
	p-value	*Odd ratio*	*Lower*	*Upper*
Duration of presentation (hours)	0.010	1.08	1.02	1.15
Lactate	0.026	18.81	1.42	249.24
HP	0.094	0.08	0.00	2.29

brought by attendants of patients in most patients. Patients are mostly conscious and can narrate their history. Testing for paraquat in plasma and urine was not done due to the unavailability of semi-quantitative testing during the study period though it is now available. Sodium dithionate tests (plasma and urine) can be done at bedside and can be used to assess the severity and outcome of paraquat poisoning. An early urine or plasma sample (10 mL) in an alkaline medium (5 mL of 1 molar sodium hydroxide) can confirm the presence of paraquat (blue urine) when a spatula of sodium dithionite reagent is added to the test tube. The darker the color the higher the concentration of paraquat. It is indicative of confirmation of diagnosis and severity. These tests are mostly unavailable and hence have not been found to be useful in treatment planning or describing outcomes.[12]

Another predictor of severity is the severity index of paraquat poisoning (SIPP) score. The formula is time from paraquat ingestion to treatment) in hours × paraquat levels in plasma at admission (mg/dL). It is useful in planning as well as comparing results during treatment.[13]

Management includes early gastric lavage with adsorbents (activated charcoal 1–2 gm/kg), oxygenation and ventilation, HD, continuous venovenous hemofiltration (CVVHF) or HP along with supportive care. Patients are also given systemic steroids, cyclophosphamide, and antioxidants with the intent to reduce free radical damage Similar to our study other studies have also shown that antioxidants (Vitamin C, N acetylcysteine, Vitamin E, Edaravone) and immune suppressants such as steroids or Cyclophosphamide did not improve survival.[8,11] Overall, RRT in our study did not have significant relation with survival. Hemoperfusion was started in 12 patients who presented early, within the time window of four hours and it was observed that HP was associated significantly with improved outcome. Li et al. also observed in their study that treatment with CVVHF and HP improved survival.[14]

In another study, early HD combined with CHDF within 4 hours has been reported to be effective.[15] As paraquat strongly binds to tissues within a few hours of intake and later it is redistributed from the tissues to circulation, there is evidence of the benefit of early HP only in small to moderate amounts of paraquat intake.[10]

In a recent article it has been suggested that for better outcome, seven HP sessions should be done, starting within 4 hours of ingestion aiming to keep a target plasma concentration less than 0.2 mg/L. Charcoal-based cartridges or toxin-specific cartridges can be used for HP.[10]

Despite all these treatment measures, mortality with this Poisoning is exorbitantly high, and stringent measures need to be taken to either enforce complete prohibition as has been done in many countries with other safer alternatives as herbicides or to make its availability restricted to licensed chemicals. A study from Korea showed a decrease in mortality after the prohibition of paraquat production and sales.[16]Another phenomenon was reported, that a ban on paraquat led to an increase in consumption of other poisons putting emphasis on the fact that nations should work to decrease the motives for suicide too.[17]

Paraquat is largely available in highly concentrated form (20–50%) but the formulations used in the field are 0.07–0.14%. So, if dilute concentration is made available commercially, it might reduce the dose of poison when ingested. Some other restrictions that can be considered are that only a certified person is allowed to use, not to be stored in any food type container, and not allowed to be stored around home gardens, schools, recreational parks, playgrounds, large warning labels, different structures of paraquat containers than commonly used shapes of syrup containers. Less toxic paraquat Alternatives can be considered including glyphosate, dicamba, diquat, etc.[18] Paraquat is 28 times more toxic than glyphosate, and diquat also forms free radicals damaging the tissues but it does not have a special affinity for the lungs. Currently, there are no regulations against the use of paraquat in India, and it remains widely used in many states. In India, paraquat is marketed as a 24% concentrated solution and is approved by the Central Insecticide Board and Registration Committee. Though initially approved for nine crops in use is extended to at least 25 crops in the study area as reported in a study.[19] Though farmers were aware of its toxic nature still they don't take precautions while spraying or storing it. Strict policy formation is needed to curb this menace and governments need to take firm initiatives. States like Odisha and Kerala have taken steps and banned the use of paraquat in their states and adopted other less toxic alternatives.[20,21]

Strengths and Limitations

In this study, data was collected from a large number of patients and we were able to describe the spectrum of presentation, clinical features, organ failure, and outcome. Limitations of the study were that as it was a retrospective study, some of the information was not available in the file records. Another limitation could be that in this study we couldn't analyze the reason behind suicide attempts or accidental ingestion because of scarce information in records.

Conclusion

Paraquat is a commonly used herbicide and its ingestion results in high morbidity and mortality. Early presentation and early initiation of HP may improve the prognosis. Studies have suggested that

HP may be of benefit if the patient presents within four hours of ingestion of paraquat, still, more data is needed to support this. Many countries have prohibited the use of this pesticide and strict policy measures in India are needed to raise awareness and limit or prohibit its use in agriculture.

Clinical Significance

Paraquat Poisoning has a very high mortality worldwide. The study highlights the importance of early presentation with treatment aiming at decreasing absorption or increasing the elimination of paraquat. Hemoperfusion when started early within four hours may improve the outcome. Patients presenting with metabolic acidosis, renal failure, and respiratory distress warranting invasive or NIV had poor prognosis. The mental health, stressors, and reasons for drastic action should be sought so that remedial actions can be taken at an earlier time. Alternative less toxic herbicides and strict government regulations may hold the key to decreasing accidental or intentional intake.

Presentation at a Meeting

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