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TO ASSESS THE CLINICAL PROFILE, TREATMENT, PROGNOSIS, AND OUTCOME OF ACUTE PARAQUAT POISONING AMONG PATIENTS ADMITTED IN A TERTIARY CARE HOSPITAL, BIMS BELAGAVI: A RETROSPECTIVE STUDY

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ABSTRACT

Introduction: Paraquat is a highly toxic bipyridyl herbicide associated with significant morbidity and mortality following acute poisoning, particularly in developing countries where its availability remains widespread. Owing to the absence of a specific antidote and the rapid progression to multiorgan dysfunction, outcomes in paraquat poisoning are often poor despite intensive supportive care. Identification of clinical and laboratory factors associated with mortality is essential for early risk stratification and management. This study was undertaken to evaluate demographic characteristics, organ dysfunction, treatment modalities, and their relationship with outcomes in patients presenting with acute paraquat poisoning.
Materials and methods: This is retrospective observational cross-sectional study conducted at a tertiary care centre, Civil Hospital BIMS, Belagavi. The study included patients aged ≥18 years with confirmed acute paraquat poisoning admitted during a one-year study period. This includes patients who were self-admitted or with witness account of paraquat exposure (ingestion, inhalational, mucosal or skin contact).
Results: Study population consisted of 40 males (76%) and 12 females (23%). Out of 52 patients 23 survived and 29 had fatal outcomes. Most patients belonged to 16-25 age group (38%) followed by 26-35 age group (23%). Hematological values were altered in 23 (55%) and normal in 29 (44%). Liver function test were altered in 20 (38%) and normal in 32 (61%). Age, gender and hemodialysis did not have any significant relation with mortality.. Hemodialysis had no relation with mortality but, 17.4% of patients who received hemodialysis survived.Majority of the patients had B/L clear lung fields 31(59.62%) followed by B/L lung opacities 9(17.31%), B/L ill defined opacities 8(15.38%) and B/L GGO 4(7.69%) respectively. Significant difference between mean values of survivors and non survivors of variables in (i)amount of paraquat consumed in ml p(0.047) (ii)derranged renal function p(0.018) (iii)age p(0.041) was noted and were significant.The mean+/-SD for survivors and non survivors was 36.74+/-44.46 and 45.34+/-38.73 respectively. The difference was statistically significant with p value 0.047.
Conclusion: There is no specifc antidote for paraquat poisoning. It is important to know the early diagnosis and prevent further absorption of poison from the gastrointestinal tract. The amount of paraquat poison consumed showed a statistically significant difference between survivors and non-survivors, supporting the dose-dependent nature of paraquat toxicity. Higher ingestion is associated with increased tissue accumulation and more severe organ dysfunction. Overall, mortality in paraquat poisoning is largely driven by the severity of systemic toxicity, particularly renal dysfunction.

KEYWORDS: Paraquat poisoning; Acute herbicide intoxication; Renal dysfunction; Mortality predictors; Tertiary care hospital.

INTRODUCTION

Paraquat is a highly toxic bipyridyl herbicide introduced in the 1950s and widely used in agriculture due to its effectiveness and low cost. Despite being classified as a restricted-use pesticide in many countries, it remains easily accessible in developing nations like India.[1] Acute paraquat poisoning, most often following intentional ingestion, carries a very high mortality rate. Once absorbed, it generates excessive reactive oxygen species leading to multiorgan

dysfunction, with the lungs being the primary target organ.[2] Patients commonly develop acute respiratory distress syndrome (ARDS), pulmonary fibrosis, acute kidney injury, hepatotoxicity, metabolic acidosis, and circulatory instability. Notably, there is no specific antidote, and reported case fatality rates range from 60–70% or higher.[3] Although various treatment strategies such as early hemoperfusion, immunosuppressive therapy, antioxidants, and supportive critical care have been attempted, outcomes remain inconsistent and standardized management protocols are lacking. Data from the Indian population, particularly regarding clinical profile, management practices, and prognostic indicators, are limited.[5] Therefore, this study aims to evaluate the clinical features, treatment modalities , also prognosis and outcomes of acute paraquat poisoning in patients admitted to a tertiary care hospital, thereby contributing to improved understanding and evidence-based management of this lethal intoxication.

MATERIAL & METHODS

Retrospective Observational cross-sectional study was conducted at tertiary care hospital (Civil Hospital BIMS, Belagavi), after ethical approval IEC no. 74/2025-26 from hospital ethical committee.
Study included patients aged 18 and above with confirmed acute paraquat poisoning during study period of 1 year.
Data was obtained manually from patients' medical records. Information recorded was age, gender, amount of paraquat consumed in ml, admission date, length of hospital stay, clinical and laboratory findings, hemodialysis and outcome.

Statistical analysis

The data was analyzed using SPSS version 20.
The collected data is presented in percentage and diagrams.
Chi-square test is used to see the association between amount of the paraquat compound consumed and the resulting clinical features and complications.
$p < 0.05$ is considered as significant.
Descriptive statistics such as mean, standard deviation, and percentage are calculated for demographic and clinical characteristics.
Mann-Whitney U test was applied to see the difference between median values of survivors & non survivors as the data was not normal.

Results

Table 1. Distribution of age of the patients with paraquat poisoning (n=52)

Age	Count	Percentage
16-25	20	38.46
26-35	12	23.08
36-45	8	15.38
46-55	8	15.38
56-65	1	1.92
66-75	3	5.77
Grand Total	**52**	

In table 1, most of the patients were belongs to 16-25 years 20(38.46%) followed by 26-35 years 12(23.08%), 36-45 years and 46-55 years 8(15.38%), 66-75 years 3(5.77%) and 56-65 years 1(1.92%) respectively.

Fig. 1: Distribution of patients with respect to age.

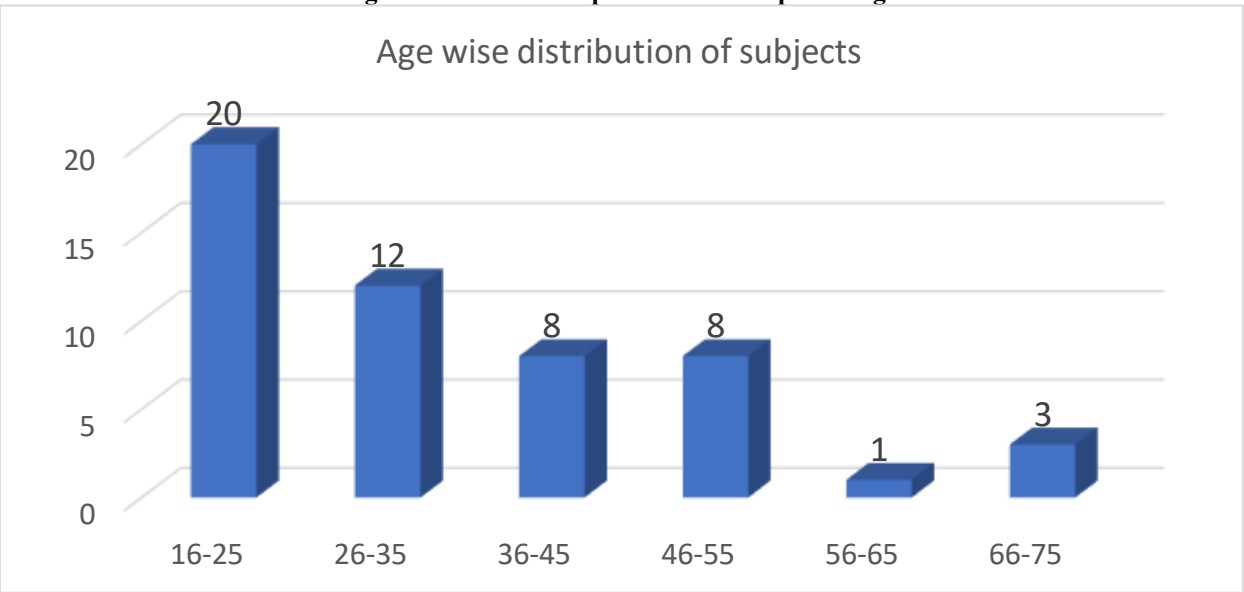

Table 2: Distribution of Gender (n=52)

Gender	Count	Percentage
F	12	23.08
M	40	76.92
Grand total	**52**	

In table 2, the majority were male (n=40, 76.92%) and female (n=12, 23.08%) were least.

Fig. 2: Distribution of patients with respect to gender.

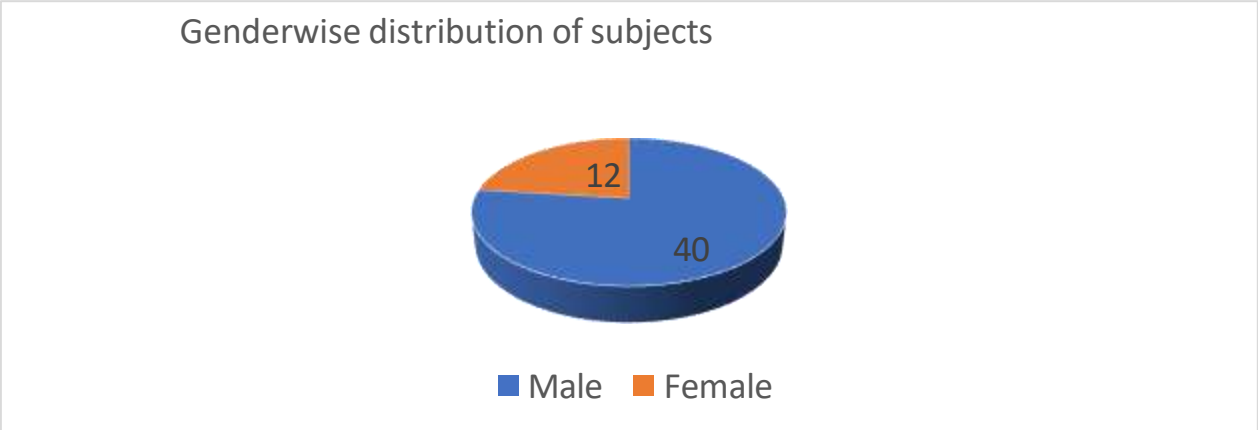

Table 3 : Comparative analysis of survivors and non survivors in different age groups

Age	Survivor	Non-survivor	Grand Total
16-25	11	9	20
26-35	6	6	12
36-45	5	3	8
46-55	0	8	8
56-65	0	1	1
66-75	1	2	3
Grand Total	**23**	**29**	**52**

Chi square=9.467 & p=0.092
There was no association between Age & outcome.

Table 4 : Comparative analysis of survivors and non survivors with respect to amount of paraquat consumed

Paraquat consumed in ml	Survivor	Non-survivor	Grand Total
5	1	0	1
10	7	3	10
15	4	1	5
20	2	4	6
30	2	9	11
50	3	6	9
60	0	1	1
80	2	2	4
100	1	2	3
200	1	1	2
Grand Total	**23**	**29**	**52**

Chi square = 11.313 & p= 0.255
There was no association between consumption of paraquat and outcome.

Table 5 : Comparative analysis of survivors and non survivors in different genders

Gender	Survivor	Non survivor	Grand Total
F	5	7	12
M	18	22	40
Grand Total	**23**	**29**	**52**

Chi square = 0.042 & p=838
There was no association between gender and outcome.

Table 6 : Clinical characteristics and outcome of paraquat intoxicated patients (n = 52)

		95% confidence interval					
Variable	Mean ±SD	Lower limit	Upper limit	Median	IQR	Test statistic	P value
Consumption of paraquat in ml							
Survivor	36.74+44.46	17.51	55.96	15	40	1.99	0.047*
Non-survivor	45.34+38.73	30.61	60.08	30	30		
Hospital days							
Survivor	5.13+5.11	2.92	7.34	3	7	0.757	0.449
Non-survivor	4.93+6.27	2.55	7.32	2	5.5		
RFT Urea							
Survivor	55.04+50.81	33.07	77.01	31	71	0.931	0.352
Non-survivor	77.65+72.55	50.05	105.24	38	101.5		
RFT creatinine							
Survivor	1.81+1.41	1.2	2.42	1.3	1.0	2.361	0.018*
Non-survivor	3.24+2.69	2.22	4.27	2.4	2.55		
Age							
Survivor	30.74+11.99	25.55	35.92	26	13	2.039	0.041*
Non-survivor	38.14+14.99	32.43	43.84	35	25		

There was a significant difference between median values of survivors & non survivors of variables consumption of paraquat (in ml), RFT creatinine & age.

Study population consisted of 40 males (76%) and 12 females (23%). Out of 52 patients 23 survived and 29 had fatal outcomes. Most patients belonged to 16-25 age group (38%) followed by 26-35 age group (23%).
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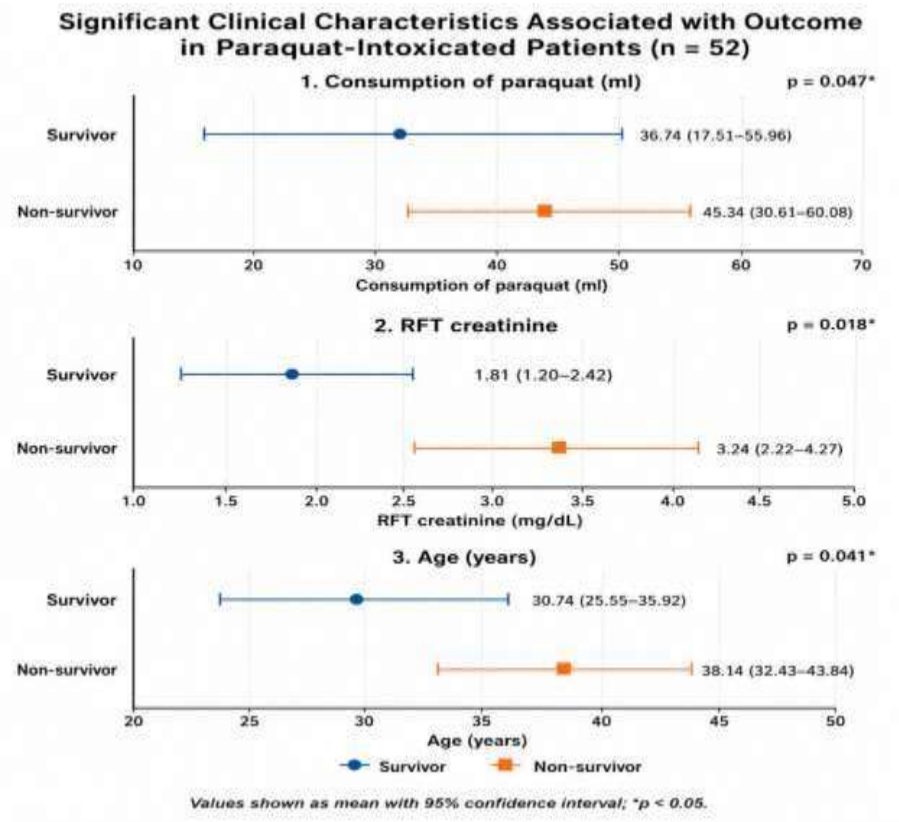
Significant difference between mean values of survivors and non survivors of variables in (i)amount of paraquat consumed in ml p(0.047) (ii)derranged renal function p(0.018) (iii)age p(0.041) was noted and were significant.
The mean+/-SD for survivors and non survivors was 36.74+/-44.46 and 45.34+/-38.73 respectively. The difference was statistically significant with p value 0.047.

Figure



DISCUSSION

Paraquat poisoning remains a highly lethal clinical entity, particularly in developing countries due to its easy availability and lack of a specific antidote. Despite advances in critical care, mortality continues to be high, reflecting the potent systemic toxicity of the compound. The present study evaluates demographic, clinical, and laboratory factors associated with outcomes in patients with acute paraquat poisoning.[4]

A male predominance and a higher incidence in younger age groups were observed, likely reflecting occupational exposure and accessibility of herbicides among young adults. However, gender did not show a significant association with mortality, suggesting that sex influences exposure patterns rather than outcomes. Although age was not an independent categorical predictor of mortality, a significant difference in mean age between survivors and non-survivors was noted, indicating that age-related physiological reserve may influence disease severity.[2]

Renal dysfunction emerged as a significant predictor of mortality in the present study. Patients with deranged renal function had poorer outcomes compared to those with preserved renal parameters. This finding is clinically relevant, as the kidneys are the primary route of paraquat elimination. Impaired renal function leads to reduced toxin clearance, prolonged systemic exposure, and worsening oxidative injury, thereby contributing to disease progression and increased mortality.[6]

The amount of paraquat consumed showed a statistically significant difference between survivors and non-survivors, supporting the dose-dependent nature of paraquat toxicity. Higher ingestion is associated with increased tissue

accumulation and more severe organ dysfunction. Although estimation of the ingested quantity is often imprecise due to reliance on patient history, this finding highlights the importance of early risk stratification based on suspected exposure volume.

After being ingested, Paraquat is rapidly distributed to organs and tissues, particularly the lungs. The lung Paraquat concentration is more than 10 times higher than that in the plasma. It has been suggested that the mechanism is primarily related to oxidative damage, reactive oxygen species, immune activation, inflammatory mediators.[8]Paraquat is excreted primarily by kidneys, and therefore, hemoperfusion has often been indicated as an appropriate step for treatment and is considered 4–6 times more effective than hemodialysis.[9]

Hemodialysis did not demonstrate a statistically significant association with mortality, although a proportion of patients who underwent hemodialysis survived. This suggests a limited supportive role rather than a definitive survival benefit. Conventional hemodialysis has restricted efficacy in removing paraquat once tissue distribution has occurred and is primarily useful for managing complications such as acute kidney injury. Overall, mortality in paraquat poisoning is largely driven by the severity of systemic toxicity, particularly renal dysfunction and toxin dose, underscoring the importance of early recognition, prompt management, and preventive strategies to reduce exposure.[7]

Across published literature, including the Vadlani VB et al., 2023 and Goyal P et al., 2024 studies, a consistent male predominance has been reported in acute paraquat poisoning, with males constituting 82.8% and 84% of cases respectively, similar to the present study where males accounted for 76% of patients, with no significant association between gender and mortality. Younger age groups formed the majority of cases across studies; however, while age was not significantly associated with outcomes in the Vadlani VB et al., 2023 and Goyal P et al., 2024 studies, the present study demonstrated a significant difference in mean age between survivors and non-survivors (36.74 ± 44.46 vs 45.34 ± 38.73 years; $p = 0.047$). Renal dysfunction emerged as a strong predictor of mortality in both the Vadlani VB et al., 2023 ($p = 0.001$) and Goyal P et al., 2024 ($p < 0.001$) studies, a finding that was similarly observed in the present study ($p = 0.018$). The requirement for hemodialysis did not show a significant survival benefit in either the Vadlani VB et al., 2023 and Goyal P et al., 2024 studies; however, in the present study, 17.4% of patients who underwent hemodialysis survived, indicating a limited supportive role. The amount of paraquat consumed showed a significant association with mortality in the present study ($p = 0.047$), supporting the dose-dependent nature of paraquat toxicity, despite variability in the strength of this association across earlier reports.

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.Alternatives can be considered including glyphosate, dicamba, diquat, etc.[10]

CONCLUSION

There is no specifc antidote for paraquat poisoning. It is important to know the early diagnosis and prevent further absorption of poison from the gastrointestinal tract. The amount of paraquat poison consumed showed a statistically significant difference between survivors and non-survivors, supporting the dose-dependent nature of paraquat toxicity. Higher ingestion is associated with increased tissue accumulation and more severe organ dysfunction. Overall, mortality in paraquat poisoning is largely driven by the severity of systemic toxicity, particularly renal dysfunction.

REFERENCES

1. Sukumar CA, Shanbhag V, Shastry AB. Paraquat: The Poison Potion. Indian J Crit Care Med 2019;23(Suppl 4):S263–S266.
2. IAR Journal of Medical Sciences. Clinical profile and outcome predictors in acute paraquat poisoning. IAR J Med & Surg Res 2023;4(5):12-16.
3. Indian Journal of Critical Care Medicine. Paraquat poisoning: clinical characteristics and predictors of mortality. Indian J Crit Care Med. 2024;28:741–747.
4. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. BMC Public Health 2007;7:357. DOI: 10.1186/1471-2458-7-357
5. Wagner SL. Clinical toxicology of agricultural chemicals. Noyes Data Corporation; 1983. p. 306.
6. Gawarammana IB, Buckley NA. Medical management of paraquat ingestion. Br J Clin Pharmacol. 2011 Nov;72(5):745-57. doi: 10.1111/j.1365-2125.2011.04026.x. PMID: 21615775; PMCID: PMC3243009.
7. Yadav M, Shah NA, Ghimire R, et al. Acute paraquat poisoning in an adolescent with compromised outcome: A case report. Sage Open Medical Case Reports. 2024;12. doi:10.1177/2050313X241240098.
8. Parvin R, Hasan K, Sarkar P, Mouri NN. Fatal Paraquat Poisoning in a 15-Year-Old Girl. J Enam Med Coll. 2017;7(2):107–10.
9. Wang JW, Yang X, Ning BY, Yang ZY, Luo LH, Xiao H, et al. The successful treatment of systemic toxic induced paraquat poisoning by skin absorption: case reports and a literature review. Int J Clin Exp Pathol. 2019;12(9):3662–70.
10. Llamas M, Edel A. Alternative pesticides. Available from: