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Case Series

Internal Medicine Section

Clinical Profile of Paraquat Poisoning from a Tertiary Care Hospital in Southern India: A Case Series

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ABSTRACT

Paraquat is a 1,1´-dimethyl-4,4´-bipyridinium dichloride herbicide widely used in agriculture. It is highly toxic even in a very minimal amount (10-20 mL), which on consumption, is associated with multiorgan failure. Intoxication may be accidental or suicidal, and the route of exposure is oral ingestion, inhalation, or transdermal absorption. Few countries have banned this compound or its use is restricted, but in developing countries like India, its unrestricted availability makes it a popular tool for deliberate self-harm. This case series pertains to an observation of 15 patients admitted to a teaching hospital with paraquat poisoning. The present case series included 15 patients, mostly males 10 (66.6%) in the age group of 18-50 years. Among 15 patients, 12 were suicidal (80%), and gastrointestinal symptoms like vomiting (100%) and difficulty swallowing (66.6%) were the most common initial presentation after intake. Renal involvement was the most common (93.3%), followed by lung (60%) and liver (60%). Patients were treated with corticosteroids, cyclophosphamide, antacids, vitamin C and chlorhexidine mouthwash. Out of total patients, 8 (53.3%) underwent haemodialysis due to acute renal failure and 10(66.6%) patients received N-Acetylcysteine due to acute liver injury. Mortality of patients with paraquat poisoning was 45.45%. Paraquat poisoning is lethal with no effective antidote. Severity of the poisoning depends on amount of compound ingested. Morbidity and mortality are high due to multiorgan failure or respiratory failure due to pulmonary fibrosis. Policymakers should focus on either banning the compound or restricting its availability due to its high toxicity.

Keywords: Corticosteroids, Herbicide, Mortality, Pulmonary fibrosis, Toxicology

INTRODUCTION

Paraquat is a commonly used herbicide in developing countries, including India. It is cheap and easily available (unregulated use), used for agricultural use by 1962 [1]. It is a pungent, corrosive liquid, commonly available in market as Gramaxone, Uniquat, Aalquit etc., It is a restricted use herbicide due to its highly toxic nature and compound primarily used for weed and grass control [2]. It is banned in many countries due to its toxicity and use with suicidal intent [3]. In India, paraquat dichloride with a concentration of 24% is available and authorised for use by the Central Insecticide Board and Registration Committee [4]. Mode of poisoning is suicidal or accidental, it's lethal even in small amounts of 10-20 mL, and mortality ranges from 50-75% [5,6]. Oral ingestion can cause corrosive injury (erosions, ulcerations and necrosis) to the gastrointestinal tract. Paraquat absorption is quick but incomplete from the gastrointestinal tract. After absorption, paraquat accumulates in alveolar type I and type II cells due to structural similarity to naturally occurring polyamines. Paraquat is secreted from kidneys and it accumulates in proximal convoluted tubular epithelial cells. Then paraquat undergoes redox cycling and generates Reactive Oxygen Species (ROS). ROS will cause lipid peroxidation, mitochondrial damage, and protein denaturation, leading to mitochondrial dysfunction and cell death [1,7]. Paraquat causes pulmonary alveolitis, lung fibrosis due to oxidative stress and fibroblast activation. Paraquat causes acute tubular necrosis in kidneys and hepatocellular injury due to oxidative damage [1]. High mortality in paraquat poisoning is due to lung fibrosis-related respiratory insufficiency and multiorgan failure.

There is no specific antidote, and most of the patients receive supportive measures like steroids, immunosuppressants, antioxidants and haemoperfusion/haemodialysis to remove the toxin from the body. Haemoperfusion is very useful in acute poisoning, significantly removing paraquat from the blood before it accumulates in tissues and improving survival. In haemoperfusion, patient's blood is circulated through a cartridge containing adsorbent materials (activated charcoal or resins). These materials have high surface area and bind with toxic substances present in blood. The filtered blood is free from toxic substances and returned to the patient's circulation. As per a study done by Hsu CW et al., early haemoperfusion (<5 hours) with repeated pulse therapy is associated with decreased mortality [8]. In a study done by Rao R et al., early haemoperfusion (<6 hours) improved survival rates [9]. In patients with lethal dose paraquat ingestion haemoperfusion may not be useful due to rapid absorption, accumulation in tissues and redistribution [1]. Haemodialysis is less effective for paraquat poisoning due to its molecular properties, and it works on the principle of diffusion. It can be used if haemoperfusion is not available or if patient's are in state of renal failure [1]. The present case series pertains to the clinical profile and outcome of paraquat poisoning presented to a tertiary case teaching hospital.

CASE SERIES

There were 15 cases of paraquat poisoning, aged 18-50 years, who were admitted to a tertiary care center of teaching hospital in Southern India the period from January 2019 to December 2020. Patients with >18 years with history of a paraquat poisoning were included in the study, patients with co-ingestions and a history of liver/kidney diseases were excluded. Paraquat poisoning diagnosis is confirmed by history, examination and image of container shown by attendees in their mobile or paraquat container brought by attendees. Data was collected retrospectively from the patients hospital records after obtaining approval from the ethical committee (IEC study no.181/2024). The patient information, such as age, gender, quantity ingested, presenting symptoms, organ systems involved, laboratory reports, and outcome were collected. All quantitative data is expressed as percentages.

Demographic profile and clinical presentation are shown in [Table/Fig-1]. Male patients 10 (66.6%) were more than female 5 (33.3%) who had taken the compound, the age group was between 18-50 years of age. Out of the 15 patients with poisoning, 12 (80%) were suicidal and 3 (20%) were accidental. Gastrointestinal symptoms

Case no.	Age (years)	Gender	Manner of poisoning	Quantity	Gastrointestinal system	Respiratory system
1	35	Female	Accidental	Unknown	Vomiting, dysphagia, oral ulcers	No
2	50	Male	Accidental	15 mL	Vomiting, hematemesis, oral ulcer s, dysphagia	No
3	30	Male	DSH*	300 mL	Vomiting, abdominal pain	No
4	18	Male	DSH	20 mL	Vomiting	No
5	23	Male	DSH	50 mL	Vomiting, oral ulcers, dysphagia	No
6	18	Male	DSH	5 mL	Vomiting, abdomen pain	No
7	24	Male	Accidental	Unknown	Vomiting, dysphagia, oral ulcers	No
8	32	Male	DSH	10 mL	Vomiting, dysphagia, abdomen pain	No
9	35	Female	DSH	20 mL	Vomiting, dysphagia	Dyspnea
10	20	Male	DSH	Unknown	Vomiting	No
11	25	Male	DSH	Unknown	Vomiting, abdomen pain	Dyspnea
12	32	Female	DSH	50 mL	Vomiting, dysphagia	Dyspnea
13	30	Male	DSH	15 mL	Dysphagia, vomiting, haematemesis, oral ulcers	No
14	25	Female	DSH	30 mL	Vomiting, dysphagia, abdomen pain	Dyspnea
15	24	Female	DSH	100 mL	Vomiting, dysphagia	Dyspnea

[Table/Fig-1]: Demographic profile and clinical presentation (*DSH: Deliberate Self-Harm).

such as vomiting 15 (100%), dysphagia/oral ulcers 10 (66.6%), abdomen pain 5 (33.3%), haematemesis 2 (13.3%) were the most common initial presentation after compound intake and presentation to Emergency Department (ED).

Investigations of study population are shown in [Table/Fig-2]. Liver involvement is seen in 9 (60%) out of 15 patients. Kidney involved in 14 patients (93.3%) out of 15. Lung involvement in the form of Acute Respiratory Distress Syndrome (ARDS), pneumonitis, and pulmonary fibrosis was seen in 9 (60%) out of 15 patients. Endoscopy done in three patients showed Zargar 2A grading of esophageal injuries. One patient (6.6%) had a seizure during the hospital stay [10].

(26.6%) were Discharged Against Medical Advice (DAMA). Mortality of patients with paraquat poisoning was 45.45% (excluding DAMA). Among the patients who survived, the amount of paraquat ingested was <20 mL and multiple organs were not involved. Predictors of mortality were the amount of paraquat ingested, pulmonary involvement, and multiorgan failure. Out of the patients who survived, three patients followed up after two weeks, their creatinine, LFT, and oxygen saturation were found to be normal. One patient followed up after one week, was on oxygen by face mask to maintain oxygen saturation between 88-90%, and creatinine was 3.2 mg/dL with good urine output. One patient did not come for follow-up.

Case no.	Creatinine (mg/dL)	Urea (mg/dL)	TB (g/dL)	AST (U/L)	ALT (U/L)	ALP (U/L)	PT (Seconds) /INR	Respiratory system (x-ray /CT chest)	Oesophageal injury (Endoscopy)
1	2.78	92	0.62	25	33	85	16.6/1.2	No	Zargar 2A
2	3.02	120	0.63	31	27	158	12.5/1.0	No	Not done
3	7.06	55.6	3.30	260	80	94	12/1.24	No	Not done
4	5.07	143	0.62	14	20	172	13.1/1.17	No	Zargar 2A
5	7.12	94	6.63	712	1935	86	29.1/2.60	ARDS	Not done
6	1.41	66	0.96	28	42	60	13/1.2	No	Not done
7	11.56	132	5.92	119	179	205	12.7/1.1	Pulmonary Fibrosis	Not done
8	7.64	134	2.24	37	28	112	14.8/1.3	ARDS	Not done
9	5.21	177	3.58	138	308	425	17.3/1.55	Pulmonary fibrosis	Not done
10	2.88	129	3.24	59	93	243	26/2.34	Pneumonitis	Zargar 2A
11	18.09	237	8.48	456	409	190	13.6/1.18	Pneumonitis	Not done
12	7.34	156	5.49	145	192	147	15/1.30	No	Not done
13	2.79	83	0.95	37	73	94	13/1.16	Pneumonitis	Not done
14	2.10	92	0.22	18	16	58	15.8/1.41	Pneumonitis	Not done
15	0.95	36	3.69	29	103	198	13/1.13	Pulmonary Fibrosis	Not done

[Table/Fig-2]: Investigations (All the values mentioned in the table are peak levels during hospital stay).
TB: Total bilirubin; AST: Aspartate transaminase; ALT: Alanine transaminase; ALP: Alkaline phosphatase/INR: prothrombin time/International normalised ratio; CT chest: Computed tomography chest

Organ systems involved, treatment, and outcomes of paraquat poisoning patients are shown in [Table/Fig-3]. Patients were treated with corticosteroids (Inj. Dexamethasone 8 mg i.v. TID for 3-5 days or Inj. Methylprednisolone 1 gram i.v. OD for three days), Inj. Cyclophosphamide 15 mg/kg i.v. OD for three days, antacids (Inj. Pantoprazole 40 mg i.v.), Vitamin C 500 mg TID and chlorhexidine mouth wash. 8 (53.3%) patients underwent haemodialysis due to acute renal failure and 10 (66.6%) patients received N-acetylcysteine (150 mg/kg over one hour, followed by 12.5 mg/kg over four hours and 6.25 mg/kg for 67 hours) due to acute liver injury. Haemoperfusion was not done for any patient. Out of 15 patients, 6 (40%) got discharged, 5 (33.3%) died, and 4

DISCUSSION

Paraquat is a dipyridyl compound that causes direct cellular damage by the production of superoxide radicals or other ROS and nitrite radicals. After ingestion, the greatest paraquat concentration is found in the lungs, and the concentration peaks in five to seven hours. Primary target of paraquat is the lung and kidney [1]. It has structural similarity to polyamines and accumulates in alveolar cells. It is secreted by the kidney and accumulates in proximal tubular cells [1]. Liver injury is due to mitochondrial damage and endoplasmic reticulum degranulation [11]. Modes of exposure to paraquat are ingestion, inhalation and dermal exposure. Diagnosis is mainly by

Case No.	Age (years)	Quantity	Organ systems involved				Treatment received					Outcome
			Liver	AKI*	ALI**	GI+	Steroids	Cyclophosphamide	N acetyl cysteine	Haemodialysis	Antacids	
1	35	Unknown	No	Yes	No	Yes	Yes	No	No	No	Yes	Discharged
2	50	15 mL	No	Yes	No	Yes	No	No	No	No	Yes	Discharged
3	30	300 mL	Yes	Yes	No	Yes	Yes	No	Yes	Yes	Yes	Died
4	18	20 mL	No	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Discharged
5	23	50 mL	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Died
6	18	5 mL	No	Yes	No	Yes	Yes	Yes	Yes	No	Yes	Discharged
7	24	Unknown	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	Discharged
8	32	10 mL	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	DAMA
9	35	20 mL	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	DAMA
10	20	Unknown	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	DAMA
11	25	Unknown	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	Died
12	32	50 mL	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	Died
13	30	15 mL	No	Yes	Yes	Yes	Yes	No	No	No	Yes	Discharged
14	25	30 mL	No	Yes	Yes	Yes	Yes	No	No	No	Yes	DAMA
15	24	100 mL	Yes	No	Yes	Yes	Yes	No	Yes	No	Yes	Died

[Table/Fig-3]: Organ systems involved, treatment, and outcomes of paraquat poisoning patients.
*AKI: Acute kidney injury; **ALI: Acute lung injury; +GI: Gastrointestinal; DAMA: Discharged against medical advice

history of exposure and amount of ingested. Lethal dose is >40 mg/kg body weight. Simple bedside test such as plasma/urine sodium dithionate test can be used to assess systemic paraquat toxicity [1]. No specific antidote is available and there are no evidence-based recommendations for use of antioxidants or immunosuppressants. Management is supportive, gastric decontamination with activated charcoal should be done in patients presenting within 1-2 hours and gastric lavage should not be done. Routine oxygen shouldn't be administered due to production of ROS [7]. Predictors of mortality and morbidity following paraquat poisoning are high creatinine [12], amount of paraquat ingested, pulmonary and cardiovascular system involvement [13], high creatinine, hypokalemia and Multi-Organ Dysfunction Syndrome (MODS) [14].

In the present case series of 15 patients, most were males 10(66.6%) in the age group of 18-50 years. Gastrointestinal symptoms such as vomiting (100%), dysphagia/oral ulcers (66.6%), abdominal pain (33.3%) were the predominant initial symptoms after compound intake. Hepatic involvement was seen in 60% of the present study population. Hepatic involvement was seen in 47% of patients in a study done by Narendra SS and Vinaykumar S and 58.3% in a study done by Sahu MR et al., [15,16].

Acute Kidney Injury (AKI) was seen in 93% of patients. AKI was seen in 81.8% of patients in a study done Ravichandran R et al., 83.3% in a study done by Sahu MR et al., and 78.5% in a study done by Kanchan T et al., [5,16,17]. Patients with AKI are associated with a significant increase in mortality when compared to those without AKI [18]. AKI further complicates the paraquat clearance from the body as it is primarily excreted via kidneys.

Lung injury was seen in 60% of our study patients. Lung injury was seen in 61.8% of patients in a study done by Ravichandran R et al., 53.3% in a study done by Narendra SS and Vinaykumar S and 91.6% in a study done by Sahu MR et al., [5,15,16]. Initially, diffuse alveolar damage occurs and with increasing concentration of reactive oxygen radicals in the pulmonary tissue, which is a result of active cellular uptake and high oxygen concentration, there is widespread damage ultimately culminating in pulmonary fibrosis [19].

The mortality rate in the present study population was 45.45%. Mortality rate was 72.7% of patients in a study done by Ravichandra R et al., 61% in a study done by Rao R et al., 21.8% in a study done by Tajai P et al., and 91.6% in a study done by Sahu MR et al., [5,9,13,16].

CONCLUSION(S)

Acute poisoning from paraquat is a concern due to high morbidity and mortality, and hence the study is undertaken to gather the clinical data and outcome of paraquat poisoning in the region. The study identified that deliberates self-harm is the most common manner of paraquat poisoning and is predominantly seen in young males. The study also revealed that paraquat toxicity involves multiple organ systems, and the amount of compound ingested, pulmonary involvement and multiorgan failure are the predictors of mortality. Our study showed a mortality rate of 45.45%. There are no effective antidotes and evidence-based guidelines for the management of paraquat toxicity. Due to the toxic nature of the compound and high mortality, it's important that the public should be educated about the consequences of compound ingestion and physicians should be aware of multiorgan involvement of the toxic compound and treatment modalities. Because of high mortality, paraquat compound should be banned, or sales should be restricted to license holders, and preventive measures such as avoiding using the compound, storing it in a safe place in a container, and wearing protective equipment if handling paraquat compound should be taken.

REFERENCES

[1] Sukumar CA, Shanbhag V, Shastry AB. Paraquat: The poison potion. Indian J Crit Care Med. 2019;23(Suppl 4):S263-S266.
[2] Wesseling C, Corriols M, Bravo V. Acute pesticide poisoning and pesticide registration in Central America. Toxicol Appl Pharmacol. 2005;207(2 Suppl):697-705.
[3] Watts M. Paraquat monograph. Pesticide Action Network Asia and the Pacific (PAN AP); 2011.
[4] Central Insecticide Board and Registration Committee, Directorate of Plant Protection, Quarantine and Storage. Department of Agriculture and Cooperation, Ministry of Agriculture, Government of India. Insecticides/Pesticides Registered under Section 9(3) of the Insecticides Act, 1968 for use in India as on 31.12.2018.
[5] Ravichandran R, Amalnath D, Shaha KK, Srinivas BH. Paraquat poisoning: A retrospective study of 55 patients from a tertiary care center in Southern India. Indian J Crit Care Med. 2020;24(3):155-59.
[6] Wilks MF, Fernando R, Ariyananda PL, Eddleston M, Berry DJ, Tomenson JA, et al. Improvement in survival after paraquat ingestion following introduction of a new formulation in Sri Lanka. PLOS Medicine. 2008;5(2):e49. Doi: 10.1371/journal.pmed.0050049.
[7] Chandra A, Shah KA, Mahato S, et al. Paraquat poisoning. BMJ Case Rep. 2021;14:e246585. Doi: 10.1136/bcr-2021-246585.
[8] Hsu C-W, Lin J-L, Lin-Tan D-T, Chen K-H, Yen T-H. Early haemoperfusion may improve survival of severely paraquat-poisoned patients. PLoS ONE. 2012;7(10):e48397.
[9] Rao R, Bhat R, Pathadka S, Chenji SK, Dsouza S. Golden hours in severe paraquat poisoning-the role of early haemoperfusion therapy. J Clin of Diagn Res. 2017;11(2):OC06OC08. Available from: https://www.doi.org/10.7860/JCDR/2017/24764/9166.

[10] Zargar, Showkat Ali, et al. "Ingestion of corrosive acids: Spectrum of injury to upper gastrointestinal tract and natural history." Gastroenterology. 1989;97(3)702-707.
[11] Lock EA, Paraquat Wilks MF. In: Handbook of pesticide toxicology. 3rd ed., Krieger RI San Diego: Academic Press; 2010.
[12] Gheshlaghi F, Haghirzavareh J, Wong A, Golshiri P, Gheshlaghi S, Eizadi-Mood N. Prediction of mortality and morbidity following paraquat poisoning based on trend of liver and kidney injury. BMC Pharmacol Toxicol. 2022;23(1):67. Doi: 10.1186/s40360-022-00609-y.
[13] Tajai P, Kornjirakasemsan A. Predicting mortality in paraquat poisoning through clinical findings, with a focus on pulmonary and cardiovascular system disorders. J of Pharm Policy and Pract. 2023;16:123. https://doi.org/10.1186/s40545-023-00635-z.
[14] Wilson W, Bhat R, Angadi B, Lekha N, Balaji B, Balakrishnan J. Predictors of mortality in paraquat poisoning: A two-year retrospective analysis from a tertiary care teaching hospital in South India. Indian Journal of Forensic Medicine and Toxicology. 2021;15(3):4435-43.
[15] Narendra SS, Vinaykumar S. Paraquat poisoning: A case series in South India. Int J Sci Res. 2015;4:561-64.
[16] Sahu MR, Sharma M, Rath B, Joseph B, Joseph T, Padhy KS. Clinical and pathological profile of paraquat poisoning cases- A cross-sectional study in Odisha, India. Indian J Forensic Community Med. 2020;7(4):210-15.
[17] Kanchan T, Bakkannavar SM, Acharya PR. Paraquat poisoning: Analysis of an uncommon cause of fatal poisoning from Manipal, South India. Toxicol Int. 2015;22:30-34.
[18] Weng CH, Chen HH, Hu CC, Huang WH, Hsu CW, Fu JF, et al. Predictors of acute kidney injury after paraquat intoxication. Oncotarget. 2017;8(31):51345-54. Doi: 10.18632/oncotarget.17975. PMID: 28881652; PMCID: PMC5584253.
[19] Honore P, Hantson P, Fauville JP, Peeters A, Manieu P. Paraquat poisoning "state of the art". Acta Clinica Belgica. 1994;49(5):220-28.

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