

# **Original Research Paper**

**General Medicine** 

# CLINICAL PROFILE OF PARAQUAT POISOINING IN A TERTIARY HOSPITAL

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ABSTRACT The present study is done on parquat poisoning, its modes of presentation, clinical features, complications if any and outcome. The study is conducted over the period of one year and 8 cases of paraquat consumption was studied retrospectively from the inpatient case records. Equal number of females and males were observed in the present study. The reason for consumption was suicidal in 87.5% cases. The most common presenting symptom was nausea and vomiting. The quantity ingested is one of the most factor in determining the outcome of paraquat poisoning as per our study. Five out of the eight patients died in our study. All modalities of treatment were ineffective in preventing multiorgan dysfunction syndrome. So it is very important to take measures to restrict the availability of paraquat in the open market to public.

# KEYWORDS : Paraquat poisoing , multi organ dysfunction syndrome.

AIMS AND OBJECTIVES – To study the association between clinical and laboratory parameters and outcome in patients with paraquat consumption.

**MATERIALS AND METHODS**- Present study includes patients admitted with paraquat poisioning over last one year. Analysis of data was statistically analysed in a descriptive pattern using the inpatient records. For each patient clinical information is recorded as per preformed table , which includes age, sex, quantity of paraquat consumed, clinical features which includes hepatic renal and lung complications and supportive treatment given, number of days in ICU, and outcome.

**Study design**-All case records were identified from inpatient MRD, with the diagnosis of paraquat consumption, over the last one year. For each case taken, clinical information was recorded from case sheet.

**Implications of study**-To identify the possible complications of paraquat, and help the clinicians intervene early.

#### INTRODUCTION

Paraguat dichloride (methyl viologen: PQ) is a herbicide sprayed to eliminate weeds(1). Paraguat poisoining has been increasing over the last decades. It exerts its herbicidal activity by inhibiting reduction of NADP to NADPH during photosynthesis.<sup>(3)</sup>. This leads to formation of superoxide anions, singlet oxygen, reactive oxygen species (ROS) which play an important role in cell death. It is used in the horticulture by the trade name of "Gramoxone "in India. Paraguat poisoning is difficult to manage due to lack of effective treatments to be used in humans. When consumed orally, paraquat is sequestered in the lungs and causes a release of superoxide anions which are responsible for membrane lesions and secondary necrosis of digestive tract, renal tubules, the liver and eventually pulmonary fibrosis<sup>(4)</sup>. Paraquat is rapidly but incompletely absorbed and then eliminated unchanged in urine within 12-24 hours. Clinical features are largely due to intracellular effects. Paraquat is actively taken up against a concentration gradient into lung tissue leading to pneumonitis and lung fibrosis<sup>(5)</sup> Paraquat also causes renal and liver injury. Plasma paraquat concentrations, urine and plasma dithionite tests and clinical features provide a good guide to prognosis.(6)

The very high case fatality of paraquat poisoining is due to both to its inherent toxicity and lack of any effective treatment<sup>(7)</sup> There is clinically no proven antidote for the paraquat poisoining .Activated charcoal and Fuller's earth are routinely given to minimize further absorption. Elimination methods such as hemodialysis and hemoperfusion are unlikely to change the clinical course. Immunosuppression with cyclophosphamide and methylp rednisolone and antioxidants such as acetylcysteine for free radical

scavenging have been tried. In contrast use of oxygen is detrimental in hypoxic patients as it provides more electron acceptors to increase pulmonary toxicity<sup>(8)</sup>. Therefore the most important step in treatment of paraquat poisoining should focus on decontamination as the intial absorption takes place in the first few hours of ingestion.

## METHODOLOGY

It was a retrospective study conducted in the department of medicine in a tertiary institution. The cases were analysed from in patient case records over the last one year. The study was approved by the ethical and research committee of our institution. A predesigned structured proforma was used to record the pateints demographics, symtoms and related complications and outcome.

## **RESULTS AND ANALYSIS**

The patient characteristics are depicted in the below table 1. Most of our pateints are in the young age group between 20 to 30 and between 30 to 40, indicating young age group as more common and intention being suicidal (87.5%) Among the cases analysed 4 were male and 4 were female pateints. The quantity of ingested paraquat was in 37.5% cases less than 60 ml whereas in the rest 62.5% it was above 60 ml.The quantity of paraquat consumed was very important factor to determine the consequences and a predictor of survival as per our study. Due to lack of facility, plasma paraquat levels were not done in our study. Four pateints were admitted within 6-24 hours of consumption. (50%) Rest of the pateints either reported late or received treatment from local doctors.

The commonest symptom at the time of presentation was vomiting (100%) . In 37.5% of the cases the presentation included acute kidney injury, acute liver failure and respiratory failure. Only 2 cases (25%) had no complications because of the small quantity of the poison consumed. One patient had only respiratory failure (12.5%) while one patient presented with hepatic failure and kidney injury (12.5%) while one patient presented with only acute kidney injury (12.5%). Three out of the eight patients (37.5%) received renal replacement therapy as dialysis support as shown in FIGURE 1 and FIGURE 2. The indications for dialysis was rising serum creatinine > 2.5 mg/dl and severe metabolic acidosis. Four pateints had respiratory distress ( with an average SpO2 of 60% ) and received mechanical ventilation(50%).ICU stay was prolonged more than 3 days in 25% pateints. Five out of the eight pateints died in our study with the mortality of 62.5% as shown in FIGURE 3. The three pateints survived in our study had consumed very neglible dose of the poison . The TABLE 3 survival significance shows an significant p value with the amount ingested . The quantity ingested is one of the most important factor in determing the outcome of paragaut poisoning as per our study.

## VOLUME-6, ISSUE-10, OCTOBER-2017 • ISSN No 2277 - 8160

#### IF : 4.547 | IC Value 80.26

## TABLE 1. PATIENT CHARACTERISTICS

		Count	Column N %
AGE	20 – 30	4	50.0%
	Above 30	4	50.0%
	Total	8	100.0%
SEX	F	4	50.0%
	М	4	50.0%
	Total	8	100.0%
QUANTITY	<60ml	3	37.5%
	60 - 100ml	5	62.5%
	Total	8	100.0%
INTENT	MISTAKEN FOR	1	12.5%
	COUGH SYRUP		
	SUICIDAL	7	87.5%
	Total	8	100.0%
TIME OF	6 - 24hrs	4	50.0%
PRESENTATION IN	More than 24hrs	4	50.0%
HOURS	Total	8	100.0%
COMPLICATIONS	AKI	1	12.5%
	AKI,ACUTE LIVER FAILURE	1	12.5%

	AKI, ACUTE LIVER	3	37.5%
	FAILURE,		
	RESPIRATORY		
	FAILURE		
	NIL	2	25.0%
	RESPIRATORY	1	12.5%
	FAILURE		
	Total	8	100.0%
RENAL REPLACEMENT	NIL	5	62.5%
THERAPY	Yes	3	37.5%
	Total	8	100.0%
MECHANICAL	NO	4	50.0%
VENTILATION	Yes	4	50.0%
	Total	8	100.0%
ICU STAY IN DAYS	3 days and below	6	75.0%
	Above 3 days	2	25.0%
	Total	8	100.0%
SURVIVAL	Death	5	62.5%
	Survived	3	37.5%
	Total	8	100.0%

# TABLE 2 PATEINT CHARACTERISTICS AND SURVIVAL OUTCOME

		SURVIVAL							
		Death Survived		Total					
		Count	Column N %	Row N %	Count	Column N %	Row N %	Count	Column N %
AGE	20 - 30	3	60.0%	75.0%	1	33.3%	25.0%	4	50.0%
	Above 30	2	40.0%	50.0%	2	66.7%	50.0%	4	50.0%
	Total	5	100.0%	62.5%	3	100.0%	37.5%	8	100.0%
SEX	F	2	40.0%	50.0%	2	66.7%	50.0%	4	50.0%
	М	3	60.0%	75.0%	1	33.3%	25.0%	4	50.0%
	Total	5	100.0%	62.5%	3	100.0%	37.5%	8	100.0%
QUANTITY	<60ml	0	.0%	.0%	3	100.0%	100.0%	3	37.5%
	60 - 100ml	5	100.0%	100.0%	0	.0%	.0%	5	62.5%
	Total	5	100.0%	62.5%	3	100.0%	37.5%	8	100.0%
INTENT	MISTAKEN FOR COUGH SYRUP	1	20.0%	100.0%	0	.0%	.0%	1	12.5%
	SUICIDAL	4	80.0%	57.1%	3	100.0%	42.9%	7	87.5%
	Total	5	100.0%	62.5%	3	100.0%	37.5%	8	100.0%
TIME OF	6 - 24hrs	3	60.0%	75.0%	1	33.3%	25.0%	4	50.0%
PRESENTATION	More than 24hrs	2	40.0%	50.0%	2	66.7%	50.0%	4	50.0%
IN HOURS	Total	5	100.0%	62.5%	3	100.0%	37.5%	8	100.0%
COMPLICATION	AKI	0	.0%	.0%	1	33.3%	100.0%	1	12.5%
S	AKI,ACUTE LIVER FAILURE	1	20.0%	100.0%	0	.0%	.0%	1	12.5%
	AKI,ACUTE LIVER FAILURE, RESPIRATORY FAILURE	3	60.0%	100.0%	0	.0%	.0%	3	37.5%
	NIL	0	.0%	.0%	2	66.7%	100.0%	2	25.0%
	RESPIRATORY FAILURE	1	20.0%	100.0%	0	.0%	.0%	1	12.5%
	Total	5	100.0%	62.5%	3	100.0%	37.5%	8	100.0%
RENAL	NIL	2	40.0%	40.0%	3	100.0%	60.0%	5	62.5%
REPLACEMENT THERAPY	Yes	3	60.0%	100.0%	0	.0%	.0%	3	37.5%
	Total	5	100.0%	62.5%	3	100.0%	37.5%	8	100.0%
MECHANICAL VENTILATION	NO	1	20.0%	25.0%	3	100.0%	75.0%	4	50.0%
	Yes	4	80.0%	100.0%	0	.0%	.0%	4	50.0%
	Total	5	100.0%	62.5%	3	100.0%	37.5%	8	100.0%
ICU STAY IN	3 days and below	3	60.0%	50.0%	3	100.0%	50.0%	6	75.0%
DAYS	Above 3 days	2	40.0%	100.0%	0	.0%	.0%	2	25.0%
	Total	5	100.0%	62.5%	3	100.0%	37.5%	8	100.0%

#### TABLE 3 – SURVIVAL RATE SIGNIFICANCE

	Fishers exact test	
	p value	
AGE * SURVIVAL	.500	
SEX * SURVIVAL	.500	
QUANTITY * SURVIVAL	.018	sig
INTENT * SURVIVAL	.625	
TIME OF PRESENTATION IN HOURS * SURVIVAL	.500	
COMPLICATIONS * SURVIVAL	.091	
RENAL REPLACEMENT THERAPY * SURVIVAL	.179	
MECHANICAL VENTILATION * SURVIVAL	.071	
ICU STAY IN DAYS * SURVIVAL	.357	

#### **SERUM CREATININE LEVEL - FIGURE 1**



#### **RENAL REPLACEMENT THERAPY DONE - FIGURE 2**



#### DISCUSSION

Paraquat is a nonselective contact herbicide of great toxicological importance. Paraquat is excreted primarily by the kidneys, so hemoperfusion is an appropriate step for treatment as per Suh et al. <sup>°</sup> It is difficult to remove all the paraquat by hemoperfusion since pateints swallow several times the lethal dose, and by the time hemoperfusion is administered the paraquat is already spread to lung tissue and other vital organs and only a small amount is remained in the blood circulation . In our study 37.5% pateints received renal replacement therapy in the form of hemodialysis.

VOLUME-6, ISSUE-10, OCTOBER-2017 • ISSN No 2277 - 8160

in-hospital fatality rate in our study is 62.50% as compared to Sabzghabaee et. al <sup>10</sup>, study where the in house fatality was 55%. The difference may be due to late presentation of the patients to the hospital. The paraquat would already spread to various tissues and organs within hours of consumption. The most important significant differences between survivors and nonsurvivors with respect to patient characteristics was the amount of paraquat consumed. According to Vale et al.<sup>11</sup> mild paraquat poisoining which occurs after ingestion <20 mg of paraquat ion per kg body weight results in full recovery.

#### CONCLUSION

The quantity of paraquat consumed was the most important factor in prognosis. Intial decontamination played a very important role because of its kinetics of distribution in the body. Combination of acetylcysteine, immunosuppression and antioxidant treatment and hemodialysis have been tried as there is no specific antidote available. All modalities of treatment were ineffective in preventing multiorgan dysnfunction syndrome so it is very important to take measures to restrict the availability of paraquat in the open market to the public.

#### REFERENCES

- N-acetylcysteine enhances recovery from acute lung injury in man. A randomized, double-blind placebo- controlled clinical study. Arch Toxic 1984;55 (1):11- 5Dawson JR, Norbeck K, Anundi I.
- A prospective clinical trial of pulse therapy with glucocorticoid and cyclophosphamide in moderate to severe paraquat poisoned patients. Thorax 1996; 51h661-3 Lin JL, LeuML, Liu YC, Chen GH.
- Carvalho F . Paraquatpoisonings: mechanisms of lung toxicity, clinical features and treatment. Crit Rev Toxicol. 2008; 38:13–71.
- Blake D K, Gallegher RT, Woollen B H. Improved methods for the analysis of paraquat in biological fluids. Chromatographia. 2002;55(Suppl1): \$183.
- E A Lock, M F Wilks. Paraquat. In: Krieger, R.I. (ed.), Handbook of Pesticide Toxicology (2.ed.) (2001) pp. 1559-1603
- Eddleston M,Wilks M F, Buckley N A. Prospects for treatment of paraquat- induced lung fibrosis with immunosuppressive drugs and the need for better prediction of outcome: a systematic review. QJM. 2003;96(11)809–24.
- Gil H W , Kang M S, Yang J O, Lee, Hong S Y. Association between plasma paraquat level and outcome of paraquat poisoning in 375 paraquat poisoning patients. Clin Toxicol.(Phila) 2008; Jul 46 (6) 515–8.
- Hart T B, Nevitt A, Whitehead A. A new statistical approach to the prognostic significance of plasma paraquat concentrations. Lancet. 1984; 2: 1222–3.
- Suh GJ, Lee CC, Jo IJ, Shin SD, Lee JC, Min BG, Singer AJ. Hemoperfusion using dual pulsatile pump in paraquat poisoining. Am J Emerg Med. 2008;26(6):641-648
- Sabzghabaee AM, Eizadi- Mood N,Montazeri K, Yaraghi A, Golabi M. Fatality in paraquat poisoining. Singapore Med J.2010;(6) 496-500
  Vale JA, Meredith TJ,Buckley BM,Paraquat poisoing :clinical features and
- Vale JA, Meredith TJ, Buckley BM, Paraquat poisoing :clinical features and immediate general management. Hum Exp Toxicol. 1987;6(1):41-47.