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Paraquat Poisoning: A case series from tertiary care hospital (MIMS), Mandya, Karnataka

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Abstract

Introduction: Paraquat is a commonly used herbicide in India.It is classified as a viologen. It is a quickly acting non-selective herbicide widely used in Asia. It is highly toxic to humans and can cause multisystem involvement. No specific antidote is available for its treatment till now. Because of its toxicity, it has been forbidden in western countries.

Material and Methods: The data of 10 cases of paraquat consumption admitted in past 1 year were collected from MIMS medical record section. On admission to casualty, diagnosis of paraquat consumption was established by clinical history and documentation of paraquat compound from consumption bottle. The amount of compound consumed was documented. Detailed clinical examination was done and relevant investigations were done.Patients were classified to have renal dysfunction if the serum creatinine was above 1.2 mg/dl Acute Kidney Injury if serum creatinine increases by 1.5-1.9 times baseline or if urine output less than <0.5ml/kg/hr for 6-12 hours.

Results:A total of 10 patients were included in this study and their details have been tabulated. Eight patients were admitted within 6 hours of paraquat ingestion. The commonest symptoms were burning sensation in throat, chest region (90%) followed by vomiting (80%), oral ulceration or dysphagia (70%), altered sensorium (60%), dyspnea (50%), chest pain or palpitations (10%). Hypotension was documented in four patients. Lung injury was document in 6 patients, of the 6 patients who had undergone chest x ray 5 had infiltrates.Seven patients needed dialytic support. During hospitalization, 8 patients died and 2 survived.

Conclusion: Paraquat poisoning is life-threatening with multi-organ failure and pulmonary fibrosis which is having high fatality rate. Our study showed that in-hospital mortality rate of 80%. In the absence of effective antidote and specific treatment, paraquat continues to cause very high mortality rates and if not monitored closely for few weeks, the patient may succumb to multiorgan failure.

Keywords: Paraquat Poisoning,Pulmonary fibrosis

Introduction:

Paraquat is a commonly used herbicide in India.[1] Paraquat (N, N'- Dimethyl– 4,4' - Bipyridium Dichloride) is an organic compound with clinical formula $[(C_6H_7 N)_2]Cl_2$. It is classified as a viologen. It is a quickly acting non-selective herbicide widely used in Asia. It is highly toxic to humans and can cause multisystem involvement. No specific antidote is available for its treatment till now. Because of its toxicity, it has been forbidden in European union in 2007 and in USA, it is classified under restricted use which means it can be used only by licensed applicator.[2] Use of immunosuppressive therapy (Combination of glucocorticoids and cyclophosphamide) has been shown to be beneficial in improving survival with those patients with moderate to severe poisoning and progressive fibrosis.[3,4] Here we report our experience of treating ten patients of paraquat poisoning with immunosuppressive therapy, hemoperfusion and other supportive measures at our institute MIMS, Mandya.

Material and Methods:

The data of 10 cases of paraquat consumption admitted in past 1 year were collected from MIMS medical record section. On admission to casualty, diagnosis of paraquat consumption was established by clinical history and documentation of paraquat compound from consumption bottle. The amount of compound consumed was documented. Detailed clinical examination was done and relevant investigations were done.

Patients were classified to have renal dysfunction if the serum creatinine was above 1.2 mg/dl Acute Kidney Injury if serum creatinine increases by 1.5-1.9 times baseline or if urine output less than <0.5ml/kg/hr for 6-12 hours. Renal replacement therapy was initiated if the patient had complications such as oliguria, metabolic acidosis (pH<7.1) and fluid overload. Hepatocellular dysfunction was considered in elevated serum aminotransferases (Ten times elevation of normal upper limit) or elevated bilirubin levels more than 1.2mg/dl (with direct hyperbilirubinemia).

Patients with PaO2/FiO2 ratio of 200-300 or <200 as classified as having acute lung injury or acute respiratory distress syndrome, respectively. The use of steroids, cyclophosphamide and hemoperfusion was decided on case-to-case basis by treating units. Analysis of data was performed and presented in a descriptive pattern.

Results

A total of 10 patients were included in this study and their details have been tabulated (Table 1). Most of the patients were young malesaround the age of 25-35 years who were six in number,rest were two females of around 25-40 years and two middle aged males. Eight patients were admitted within 6 hours of paraquat ingestion. The commonest symptoms were burning

sensation in throat, chest region (90%) followed by vomiting (80%), oral ulceration or dysphagia (70%), altered sensorium (60%), dyspnea (50%), chest pain or palpitations (10%). Hypotension was documented in four patients. Lung injury was document in 6 patients, of the 6 patients who had undergone chest x ray 5 had infiltrates.Seven patients needed dialytic support. During hospitalization, 8 patients died and 2 survived.

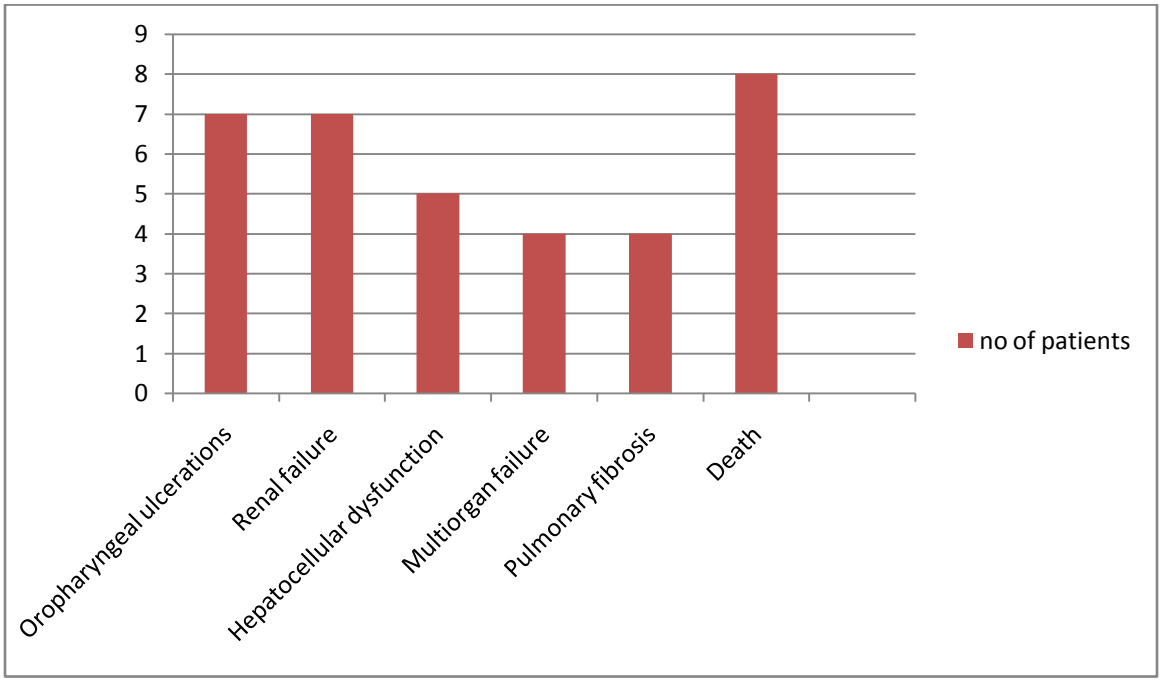
Table 1: Tabulation of cases

Particulars Of patients	1	2	3	4	5	6	7	8	9	10
AGE/SEX	31/M	41/M	29/F	51/M	22/F	43/M	19/M	32/M	20/M	30/M
Quantity Ingested (ml)	30	500	100	250	300	30	250	500	50	50

Table 2: Complications of patients

Complications	**No. of patients (%)**
Oropharyngeal ulceration	7(70%)
Renal failure	7 (70%)
Hepatocellular dysfunction	5 (50%)
Multiorgan failure	4 (40%)
Pulmomary fibrosis	4 (40%)
Death	8 (80%)

Graph 1: Commonest symptoms of patients



Discussion:

Paraquat is highly toxic to human. It belongs to group of group of dipyridyl herbicides. In plants, it disrupts photosynthesis by inhibiting the electron transport chain. It has low environmental toxicity due to rapid deactivation upon soil contact.[5] Paraquat induced toxicity is a manifestation of redox cycling and subsequent generation of reactive oxygen species.[6,7]

Other secondary effects of oxidative stress also play synergistc effect in the manifestation of overall clinical presentation of paraquat poisioning. These are lipid peroxidation,[8]mitochondrial toxicity,[9] oxidation of NADPH,[10]activation of nulear factor kappa beta,[11]and apoptosis.[12]

In this study we found that 70% of patients had renal failure which matches similar studies by R. Ravichandran et al.,[13]in which they found it to 81% . In this study we found 40 % patients had pulmonary fibrosis. Study conducted by Narendra S S et al.,[14] found to have 48% patients with pulmonary fibrosis which was similar to our study.

Patients with hepatic complications were found to be 32.8% in R. Ravichandran et al.,[13] which is similar to our study wherein we find it to be 40%. Also oropharyngeal ulcerations were seen in 70% of our patients which is similar to study done by O F Wong et al.,[15] in which it is seen in 60% patients.

Mortality rate was 80% in our study which is similar to studies done by Prasad et al.,[16] showing 83.3%, Banday et al.,[17] showing 75%, R Ravichandran et al., showing 72.7 %.[13]

Conclusion

Paraquat poisoning is life-threatening with multi-organ failure and pulmonary fibrosis which is having high fatality rate. Our study showed that in-hospital mortality rate of 80%. In the absence of effective antidote and specific treatment, paraquat continues to cause very high mortality rates and if not monitored closely for few weeks, the patient may succumb to multiorgan failure.

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