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Original Research Article

A study on clinical profile of paraquat poisoning in a tertiary care hospital

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ABSTRACT

Background: Paraquat poisoning is a weedicide used rarely for suicide. It is now being increasingly as this type of suicidal poisoning is almost always fatal as there is no specific antidote for paraquat poison. ARDS, acute kidney injury with metabolic acidosis or multi-organ failure are the frequent causes of mortality.
Methods: The study aimed to study the morbidity and mortality rates of Paraquat poisoning in tertiary care teaching hospital. Patients admitted with Paraquat poisoning were included, and the data were collected and analysed.
Results: The total number of cases admitted with paraquat poisoning in intensive care unit in three years duration were 10. All the cases were suicidal in nature. 80% of the cases had acute kidney injury with severe metabolic acidosis and 20% had mediastinitis. 70% of cases died within 48 hours of ingestion. The overall mortality rate was 100% in spite of active management.
Conclusions: Of all registered herbicides, Paraquat is the most serious and life threatening. Inspite of early haemodialysis, steroids and cyclophosphamide therapy, it was ineffective in reducing the mortality rates. Newer treatment like early hemoperfusion may help to reduce the mortality in future.

Keywords: Herbicide poisoning, Metabolic acidosis, Paraquat

INTRODUCTION

Paraquat (1, 1-dimethyl-4, 4-bipyridium dichloride) ingestion is a major cause of fatal poisoning in many parts of Asia and Pacific nations.[1] Commercial preparations of paraquat are normally sold in the form of liquid concentrate with a concentration ranging from 20% to 42%. Besides being supplied in the form of a single active ingredient, there are products in the market containing paraquat in combination with other herbicides such as sodium chlorate and 2, 4-dimethylamine. Death in paraquat poisoning is either due to significant lung injury, acute kidney injury or multi organ failure.[2] The commonest mode of poisoning with paraquat is oral intake of poison. Paraquat interferes with the intracellular electron transfer systems, thus inhibiting the reduction of NADP to NADPH (Figure 1). This will then result in the accumulation of superoxide radical which causes destruction of lipid cell membranes.[3] Toxicological analysis of plasma and urine samples is used to establish the diagnosis. Mortality rate of paraquat poisoning is directly related to plasma and urine paraquat concentrations. Paraquat is mainly eliminated by kidney and acute kidney injury is the complication of it. Ingestion of small quantities can cause severely damage to lung and kidney.[4]

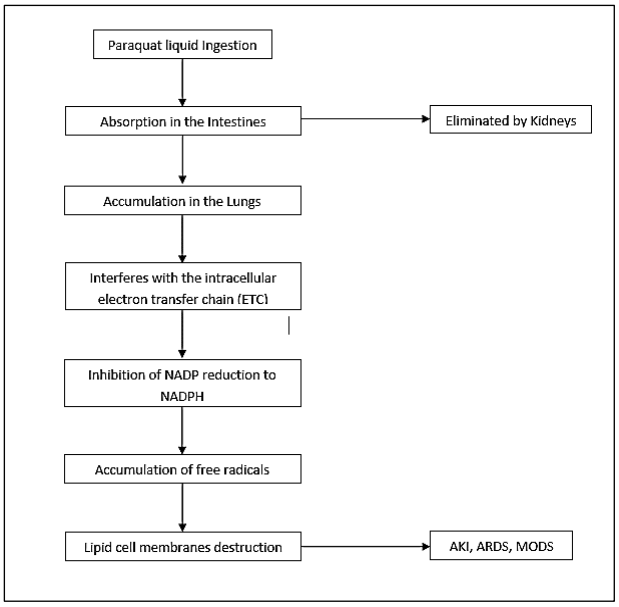

Figure 1: Pathogenesis of paraquat toxicity.

METHODS

The present study is a retrospective study conducted in a tertiary care hospital for 3 years. The case sheets and treatment charts of all the patients who were hospitalized with paraquat poisoning were reviewed. All the data were analysed anonymously. Paraquat poisoned patients who arrived at the emergency triage within 24 hours after ingestion were included in this study. All the patients who were hospitalized underwent the following tests: renal function test, liver function test, prothrombin time, arterial blood gas analysis, chest X-ray. In all the patients, the time duration between the time of ingestion of poison and commencement of therapy was noted. All the paraquat poisoning patients received treatment with 1g/kg of activated charcoal through the nasogastric tube following gastric lavage with normal saline in the emergency triage. Renal function test and liver function test were repeated every alternate day after starting therapy. Haemodialysis was initiated in all these patients. Patients who had abnormal renal function test i.e., serum creatinine > 2 mg/dl, abnormal liver function test i.e., alanine transaminase (ALT) > 80U/L and/or International Normalized Ratio (INR) > 1.5 were defined as having multiorgan failure. Patients who had PaO2 < 70 mmHg by arterial blood gas analysis at room air were defined as having acute lung injury secondary to paraquat poisoning.

RESULTS

A total of 10 patients with paraquat poisoning were included in the study. All of them were suicidal cases who had ingested liquid paraquat concentrate. The median age of the patients was 28.5 years. 80% of the paraquat poisoned patients were males (Figure 2). Only 50% of the patients were able to undergo hemofiltration. The remaining patients were managed conservatively with other supportive measures only. The study also found that only 20% of patients reached hospital within 2 hours and majority of them (60%) reached anywhere between 2-6 hours after ingestion and another 20% reached hospital after 6 hours (Figure 3).

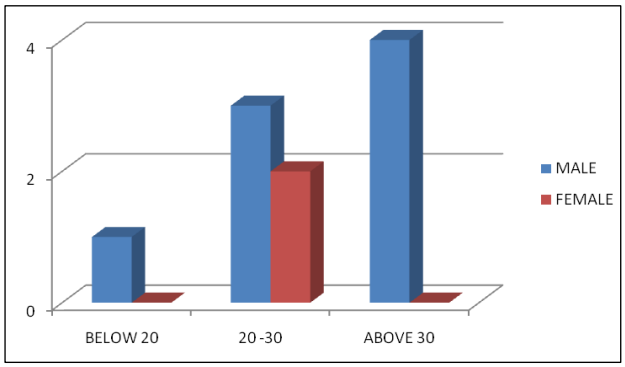

Figure 2: Demographic profile of paraquat poisoning.

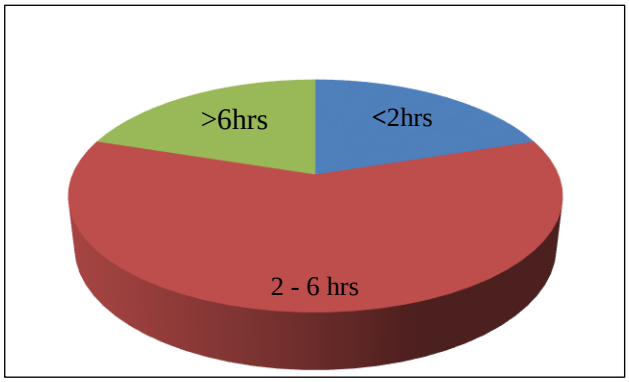

Figure 3: Distribution of time between Paraquat ingestion and reaching the hospital.

Mortality was seen in 100% of the paraquat poisoned patients in spite of intensive treatment. The majority of deaths occurred beyond 12 hours after ingestion of the paraquat compound (Figure 4).

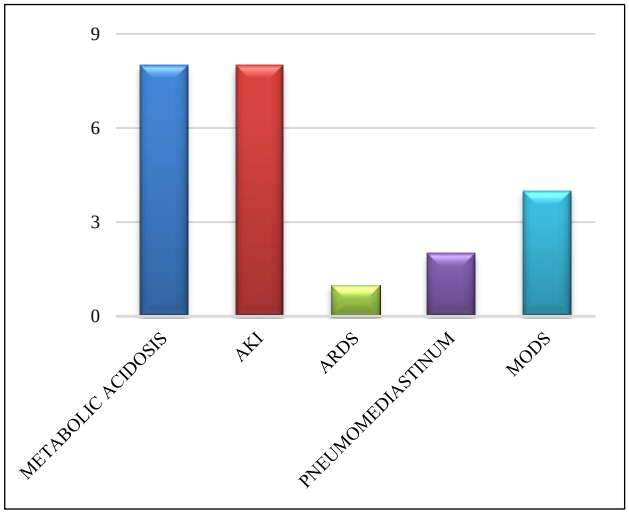

Figure 4: Complications of paraquat poisoning.

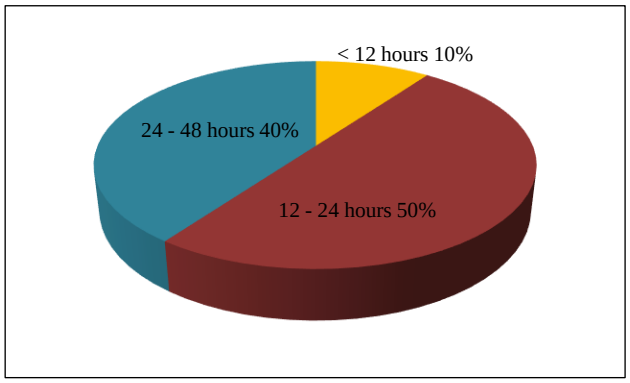

Figure 5: Distribution of time to death following ingestion.

Figure 5 shows the various complications noted in these 10 patients. 40% of patients (n = 4) died due to multi organ dysfunction syndrome and 20% of patients (n=2) died due to Acute Respiratory Distress Syndrome (ARDS). Thus, multi organ failure was the most common cause of death in patients with paraquat poisoning. 80% (n = 8) of patients developed acute kidney injury with metabolic acidosis. Out of these 8 patients, 5 of them had severe metabolic acidosis (pH < 7.2). One of the patient also developed pneumomediastinum after ingestion of the paraquat (Figure 6).

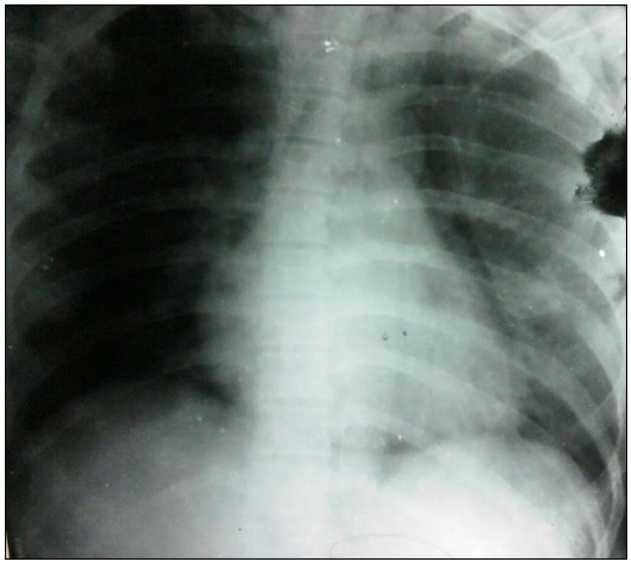

Figure 6: Chest X-ray PA view showing pneumomediastinum in the left side in the left side due to paraquat poisoning.

DISCUSSION

Paraquat poisoning is of toxicological importance in south India as it is widely used as an herbicide. The fatality rate of paraquat poisoning in our hospital was found to be 100%. In hospital cases, fatality rate ranges between 35-62% around the world.[5,6] Also majority of the patients did not reach the hospital in the golden hour due to varied reasons. This shows that in India, we still have much to do to reduce the mortality associated with paraquat poisoning.

The median age of paraquat poisoned patients was found to be 28.5 years in our study which was comparable to a study done by Kanchan et al, where the mean age was found to be about 30 years.[7] The reason for suicidal intent with paraquat poison among the youth may be due to its easy availability and its wide use as an herbicide. In our study, paraquat poisoning was more common in males than females probably reflecting the easier accessibility to the farm working population.

Treatment of paraquat poisoning is largely supportive and aimed at removing paraquat from the site of absorption. Increasing its excretion from blood and preventing pulmonary damage is the major target of management. Hospitalization is required as soon as possible in all cases of suspected paraquat poisoning.[2] Paraquat accumulates selectively in the lung tissues. Lung injury, which is mediated through lipid peroxidation, is exacerbated by the administration of oxygen therapy. Thus, supplementation of oxygen should be withheld until unless pO2 is less than 70 mm of Hg.[8] All the paraquat poisoned patients received treatment with 1g/kg of activated charcoal through the nasogastric tube following gastric lavage with normal saline in the emergency triage.

Paraquat can be removed by haemodialysis and haemoperfusion but, although the clearance values are high for paraquat compounds, the effective quantities recovered are insignificant. Prevention of death is most unlikely.[9] According to Cavalli et al, the survival rate in patients without active treatment was only 13% in non-fatal dose ingestion of paraquat poison and it increased to more than 50% with active treatment modality like haemoperfusion, in patients with fatal dose consumption.[10]

Combined therapy with haemoperfusion and Continuous VenoVenous Haemofiltration (CVVH) increased the survival duration in patients with acute paraquat poisoning.[11] CVVH as a standalone therapy was also found to be beneficial in reducing the mortality recently.[12] In a study done by Hsu et al., it was found that early haemoperfusion (within 6 hours) improved the survival outcomes in paraquat poisoned patients.[13]

Some studies found that haemoperfusion was not useful which might have been due to potentially lethal concentration of paraquat getting accumulated in highly vascular tissues of the vital organs and pneumocytes before the initiation of haemoperfusion.[14]

According to Raghavendra et al, patients who received early haemoperfusion (< 6 hours) were more likely to benefit compared to those who received late

haemoperfusion (>6 hours).[15] In the present study, only 50% of the patients who had paraquat poisoning underwent haemoperfusion due to their earlier presentation and hemodynamic stability. All of our cases developed complications, despite advanced treatment and supportive care, highlighting the high mortality rate associated with paraquat poisoning which were similar to the findings reported by Singh et al.[16]

Steroids have been tried as a measure to protect the lung however, no clear-cut benefit has been observed. There are some studies that suggested a definite trend in benefit with immunosuppressive therapy in patients with moderate to severe poisoning.[17] Superoxide dismutase, vitamins C and E, N-acetylcysteine have not yet been proven to be effective.[3,18] The prognosis of paraquat poisoning largely depends on the amount of paraquat absorbed and the time to initiate haemoperfusion after ingestion.

The amount of paraquat poison consumed was by each patient could not be accurately determined due to a combination of factors and hence all of them had to be given the doubt of fatal dose ingestion and treated aggressively. The mortality in paraquat poisoning is directly proportional to plasma and urine paraquat concentrations which couldn't be done due to non-availability of the resources.

CONCLUSION

Early diagnosis and aggressive management of paraquat poisoning is necessary. Even a less than fatal dose of paraquat poison can lead to fatal outcomes as there is no specific antidote available. Treatment remains supportive in nature. Hence it is necessary to take administrative steps to impose restriction on the availability of this fatal herbicide in the market.

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Ethical approval: The study was approved by the institutional ethics committee

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