Successful Treatment of Paraquat Poisoning In a Tertiary Care Hospital of Eastern Part of India: A Therapeutic Challenge

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I. 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 severe damage to lung and kidney.⁴

As there is no specific antidote for paraquat, the main treatment present is conservative medical management. Poison removal through digestive and circulatory system is the most frequently used treatment strategy. The extracorporeal elimination including haemoperfusion (HP), Haemodialysis (HD), Hemofiltration (HF), Plasma exchange, and continuous veno venous haemofiltration (CVVH) of which HP is more efficient in the clearance of plasma paraquat. HP was first used in 1960, initially with uncoated columns and later with charcoal.

II. Materials And Method

PATIENT 1

A 19 year old male SAMIR DHAR presented with history of ingestion of 15 ml of undiluted paraquat 48 hours prior to presentation. He complained of difficulty in deglutition, decreased urine output, and occasional vomiting. On examination he had a green coated tongue which had unhealthy mucosa and facial puffiness. There was bilateral decreased air entry in both the lungs, along with decreased urine output (<300 ml) / day. Haematological parameters showed urea 146 and creatinine 9.6 with increased Crp 86.9. Chest xray showed reticulate pattern in the basal area suggestive of early fibrosis.
HRCT thorax also suggested similar changes of fibrosis (early).
As the patient was deteriorating rapidly he was given IV Methylprednisolone with N-Acetylcysteine, followed by CHATCOALHAEMOPERFUSION (5 CYCLES).
Following which the patient was given a single dose of iv CYCLOPHOSPHAMIDE.
The patient responded well and the renal profile and pulmonary status improved considerably.
ORAL MUCOSAL LESION (BEFORE AND AFTER)
The patient was given oral vitamin E and vitamin C as an antioxidant and for better mucosal healing zinc supplement was given, and the results were remarkable.

PATIENT 2
A 17 year old female presented with history of ingestion of paraquat approximately 100ml. 3 days before admission. Initially gastric lavage was done at a local hospital, and then she was referred at medical college kolkata for management of further complications.

Her urea creatinine was deranged (180.8.5), chest xray showed presence of early fibrosis. As charcoal hemoperfusion was not available back then, she was put on hemodialysis. After 9 cycles of hemodialysis her kidney functions normalised. Along with it she was also given long term dexamethasone, and N-acetylcysteine, which showed good results.

Inhalation with N-acetylcysteine and ambroxol was done which improved the pulmonary functions considerably.

III. 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. 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. 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 male than females probably reflecting the easier accessibility to 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.

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 the pO2 is less than 70 mm of Hg. 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. According to Cavalli et al, the survival rate in patients without active treatment was only 13% in nonfatal dose ingestion of paraquat poison and
it increased to more than 50% with active treatment modality like haemoperfusion, in patients with fatal dose consumption. Combined therapy with haemoperfusion and Continuous VenoVenoous Haemofiltration (CVVH) increased the survival duration in patients with acute paraquat poisoning. CVVH as a standalone therapy was also found to be beneficial in reducing the mortality recently. 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. 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. According to Raghavendra et al, patients who received early haemoperfusion (< 6 hours) were more likely to benefit compared to those who received late.

IV. 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 there is more scope for further research; to find the perfect combination to combat this fatal toxin, to which thousands of people are losing their lives.

References

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