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Paraquat poisoning: Herbicide with fatal outcome

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***Abstract*---**Paraquat (10-dimethyl-4, 40-bipyridylium dichloride) is the most common herbicide used worldwide. Due to its inherent toxicity and lack of effective treatment, it has high case fatality of more than 50%. Paraquat exposure through ingestion or inhalation leads to multi-organ involvement including lungs, CNS, heart, kidneys and liver. Treatment is mainly supportive including initial resuscitation, gastrointestinal decontamination, anti-inflammatory, antioxidant and immunosuppressive therapy. We are reporting two cases of paraquat poisoning and their management in intensive care unit, of which one was successfully treated and discharged from ICU while other one expired.

***Keywords*---**multiorgan failure, paraquat, decontamination.

Introduction

Paraquat is one of the most widely used herbicides, discovered in 1955. It is a quick acting herbicide which inhibits reduction of NADP to NADPH during photosynthesis leading to formation of reactive oxygen species that cause destruction of plant by interacting with unsaturated lipids of membrane.[1] Poisoning occurs either through ingestion, skin contact or inhalation of the herbicide. In developing countries suicide with pesticide is a major problem with approximately 3 lakhs deaths in Asia- Pacific region.[2, 3] It has high case fatality rate due to unavailability of effective treatment and inherent toxicity. Treatment of

paraquat is supportive including antioxidant therapy, hemodialysis, hemoperfusion and immunomodulation.[4] Here in, we report two cases of paraquat poisoning which were managed in the intensive care unit.

Case Description
Case 1

A 24 year female presented in emergency department with complain of multiple episodes of vomiting, difficulty in swallowing and respiratory distress after accidental ingestion of around 10 ml of paraquat. She had history of bronchial asthma since childhood. Gastric lavage was done immediately and was shifted to intensive care unit on oxygen for observation and further management. On arrival she had heart rate of 110/min, respiratory rate- 35/min, and saturation- 98% on face mask at oxygen flow of 5 l/min. Patient was put on high frequency oxygen therapy (HFOT) at 40 litres of flow and 0.3 fraction of inspired oxygen (FiO2). On examination she was drowsy, had swollen face and tongue, bleeding oral ulcers (Figure 1), muffled voice, congestion in right eye and ronchi on chest auscultation. She was put on inj. augmentin, Candid B mouth paint, inj. pantoprazole, inj. dexamethasone, tab. vitamin C, E and inj. N- acetylcysteine. She had dysphagia most likely due to esophageal ulcers and strictures. Ryle's tube could not be inserted so total parentral nutrition was started. She gradually developed multiple organ failure including hepatitis and acute kidney injury so hemodialysis was done. Patient developed subcutaneous emphysema from neck to xiphisternum suggestive of ulceration of airway leading to air leaks. Next day she had respiratory distress, so emergency tracheostomy was done as bleeding oral ulcers and swollen tongue made the airway difficult. Patient was put on ventilator on pressure support mode. She developed cardiac arrest after two days and expired despite our best efforts to save her.

Figure 1: Image of the patient showing mouth ulcers

Case 2

A 29 year old male patient presented in emergency department with alleged history of herbicide ingestion. He complained of difficulty in swallowing of solids as well as liquids with altered behavior. He had no signs and symptoms of difficulty in breathing. Gatric lavage was done immediately and was shifted to intensive care unit. He underwent same treatment as above patient. He had

normal investigations on admission but his liver enzymes and total bilirubin increased along with kidney function tests increased progressively from next day (Figure 2). Hemodialysis was done two times during his stay in intensive care unit to support acute renal failure. He was having hallucinations so tab. olanzapine was started after psychiatric consultation. Patient improved after seven days and was shifted to ward in stable condition.

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Blood urea (mg/dl)	14	40	258	244	102	70
S.Creatinine(mg/dl)	0.8	1.2	10.1	8.0	6.2	3.2
AST (U/L)	35	109	225	232	325	219
ALT (U/L)	30	82	264	244	439	198
Total Bilirubin(mg/dl)	2.50	6.1	5.7	5.4	5.4	3.5

Figure 2. Laboratory parameters of second patient (AST- Aspartate transaminase), (ALT- Alanine transaminase)

Discussion

Paraquat poisoning occurs due to either accidental or intentional ingestion of the herbicide or through direct skin contact. On ingestion it cause symptoms like nausea, vomiting, altered sensorium, oral ulceration, difficulty in swallowing, dyspnea and abdominal pain[5] while skin exposure may cause dermatitis and burns. It is rapidly distributed in the body irrespective of the route of exposure. Systemic complications include convulsions, shock, pulmonary edema and multiorgan failure such hepatic failure, cardiac, renal and respiratory failure.[6] Outcome in paraquat poisoning depends upon the amount of herbicide ingested.[7] Treatment of parquat ingestion is its removal by gastric lavage using activated charcoal immediately on arrival in health facility, hemoperfusion within four hours of ingestion as it accumulates in lung after 5-7 hours, [5] immunomodulators such as cyclophosphamide and corticosteroids such as methylprednesolone decease inflammation so beneficial in moderate to severe cases.[8] Hemodialysis is used in acute renal failure. Principal biochemical mechanism of organ damage in paraqat is through reactive oxygen species but none of the antioxidant like N-acetyl cysteine, vitamin C and E has shown benefit in survival.[9]

Elenga et al conducted a retrospective study of 62 patients to observe the clinical features and prognosis of paraquat poisoning in French Guiana.[10] 32% patients had ulceration of oral mucosa and epigastric pain, 55% had cell disruption of liver and all (100%) had vomiting. Serum creatinine was high at admission in patients who expired. Children ingested less amount of paraquat than adults. Hemodialysis was done in one patient, 50% of the patients received corticosteroids, 71% received N-acetyl cysteine (NAC) and 58% received cyclophosphamide. However there was no difference in survival related to treatment received. Total mortality rate was 52%, with relatively high mortality in adults than children. Similar to this study our first patient who expired had higher serum creatinine levels at admission than second patient with normal serum creatinine at admission. Both patients received same treatment including hemodialysis. However first patient deteriorated, got tracheostomised for

respiratory distress and expired later while second patient improved and was discharged with stable vitals.

Conclusion

Prognosis of paraquat poisoning depends upon the amount ingested. It has high mortality rate and no specific treatment is available. Key to successful management is to establish early diagnosis, rapid removal of paraquat through lavage or hemoperfusion and to support organ failure. However more research and development should be made to find effective treatment of paraquat exposure.

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