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CASE REPORT

A Fatal Case of Paraquat Poisoning

R Hemachandar*

ABSTRACT

Paraquat (1, 1'-dimethyl-4, 4'-bipyridylium dichloride) and Diquat are popular herbicides which belong to the bipyridyl group.Paraquat is freely available in the Indian market for agricultural use. When ingested it causes oxidant free radical injury leading to renal failure, hepatotoxicity, non-cardiogenic pulmonary edema and multi organ dysfunction. There is no antidote, though immunosuppressive medications have been shown to reduce mortality in few studies. The author reports a 34 year old female admitted with suicidal paraquat poisoning who developed acute kidney injury, and Acute Respiratory Distress Syndrome (ARDS). She succumbed to her illness despite intravenous methylprednisolone and cyclophosphamide.

KEY WORDS : Paraquat, Herbicide, Acute Respiratory Distress Syndrome, Acute Kidney Injury

Introduction

Pesticide poisoning is a major global public health problem, especially in an agriculture based country like India. Herbicides are used to protect crops by controlling a wide range of weeds, thereby allowing crops to grow without competition for essential light, water and nutrients. Paraquat is widely used in agriculture as a contact herbicide. As with other pesticides, Paraquat has also been used as suicidal agent. Paraquat poisoning is associated with high mortality varying from 35% to 50%[1].Though paraquat poisoning has been reported from across the world, there are no clear recommendations for the management of patients with paraquat poisoning.

Assistant Professor, Department of Nephrology, Mahatma Gandhi Medical College and Research Institute, Puducherry, India - 607402.

***Corresponding Author**
Dr.R. Hemachandar,
Assistant Professor of Nephrology,
Mahatma Gandhi Medical College and Research Institute,
Puducherry, India – 607402,
E-mail Id: hemachandarr@gmail.com
Telephone No : +91-76675 19804

Case Report

A 34 year old female was referred to our hospital in view of oliguria, respiratory distress, and progressively increasing azotemia. Patient's husband gave history of consumption of paraquat three days prior to the admission. Immediately after taking paraquat, she developed vomiting, burning sensation in mouth, throat, and chest. Initially patient was managed in district hospital with gastric lavage and antibiotics. Next day patient developed of decrease in urine output, dry cough and breathlessness.

On examination, patient was tachypnoeic, pale and icteric. Her blood pressure was 130/90 mm of Hg, pulse rate 122 per minute, and respiratory rate 30 per minute. She had crepitations on left lower lung fields on auscultation and tender hepatomegaly. Her jugular venous pulse was not elevated. Her buccal mucosa was congested, ulcerated and edematous.

Investigations showed haemoglobin 11.2 g/dl, total leucocyte counts 7800 cells/µl, urea 186 mg/dl, creatinine 4.8 mg/dl, sodium 134 meq/L, potassium 5.6 meq/L, aspartate aminotransferase 680 U/L, alanine aminotransferase 854 U/L, total bilirubin 3.4 mg/dl and conjugated bilirubin 1.4 mg/dl. Her arterial blood gas analysis revealed PaO2 89%, pH 7.30, pCO2 20 mm of Hg, and HCO3 14 meq/l while on 60% venture mask. Chest x-ray showed bilateral parenchymal infiltrates suggestive of Acute Respiratory Distress Syndrome (Figure.No.1). Electrocardiogram showed sinus tachycardia. 2D echocardiography showed normal chambers with left ventricular ejection fraction of 61%.

She was admitted to intensive care unit and placed on mechanical ventilation after rapid sequence intubation. Patient was started on intravenous methylprednisolone 1g/day, and intravenous cyclophosphamide 750 mg/day. Next day patient was started on hemodialysis through right internal jugular catheter. Despite above efforts, patient succumbed on the third day of admission as a result of respiratory failure.

Discussion

Paraquat, a quaternary ammonium herbicide, is one of the most widely used herbicides around the world. Paraquat acts rapidly within minutes of application, allowing fields to be quickly prepared for cropping. These properties, especially broad-spectrum weed control, rapid action, and inactivation upon contact with soil, made paraquat an ideal herbicide. In India, paraquat is available as 10-20% concentrated solutions. The reason paraquat is such a commonly used suicide agent in India is due to its widespread availability, low toxic dose (10-15 ml) and relatively low cost.

In plants, paraquat acts by inhibiting photosynthesis. In humans, paraquat acts as an electron acceptor resulting in the formation of reactive oxygen species. Absorbed paraquat is sequestered in the lungs and causes release of hydrogen and superoxide anions which cause lipid damage in the cell membranes. Estimated lethal dose is just 10 - 15 ml of the solution. The World Health Organization (WHO) classified paraquat as "Moderately hazardous, class II" (WHO, 2009)[2].

Immediately after ingestion, patients develop corrosive injury to the gastrointestinal tract resulting in severe inflammation of the tongue, oral mucosa and throat pharyngeal pseudomembranes. Early signs include burning sensation in mouth and throat resulting in tharyngeal, vomiting, diarrhoea and dysphagia. Patients with severe intoxication can develop acute renal tubular necrosis, hepatocellular necrosis and acute respiratory distress syndrome. Respiratory symptoms can start within several days to several weeks after ingestion with cough, respiratory distress and pulmonary infiltrates on chest radiograph. Death may occur as late as 6 weeks after ingestion. Rarely, perforation of the oesophagus and mediastinitis may occur.

Paraquat poisoning is confirmed by adding 1 ml of urine to 1 ml of a solution of 100 mg sodium dithionite in 10 ml 1 M sodium hydroxide[3]. A blue–green colour indicates poisoning.

Conventional treatment included gastric lavage, and aggressive decontamination with fuller's earth or activated charcoal. Oxygen is relatively contraindicated early in the poisoning as it may aggravate oxygen mediated injury. Since the mode of injury is due to oxygen free radicals, immunosuppressive drugs have been tried and reported to be successful in various case reports. Intravenous methylprednisolone

15 mg/kg/day for 3 consecutive days along with intravenous cyclophosphamide 10 mg/kg/day for 2 consecutive days, followed by intravenous dexamethasone 4 mg thrice a day has been proposed for management[4]. Paraquat is not removed by dialysis and used only in patients with acute kidney injury[5].

Mortality remains high even with prompt management.Acute respiratory distress syndrome (ARDS) is the major cause of death in these patients. Patients who survive the acute poisoning or those who are exposed chronically are prone to develop restrictive lung disease due to pulmonary fibrosis.

The diagnosis in our patient was based on the history and direct verification of the container containing paraquat. Urinary examination for paraquat could not be done due to non availability. Our patient did not improve even after three doses of intravenous methylprednisolone and cyclophosphamide. The reason could be delay in referral, thereby initiation of treatment, or high dose of paraquat ingested.

Conclusion

To conclude, paraquat poisoning has become a common entity in India, yet it is rarely reported and is associated with a high mortality rate. There is no specific antidote available for paraquat poisoning. Early diagnosis and aggressive decontamination is pivotal. The role for immunosuppressive therapy in patients with paraquat poisoning is not clear due to paucity of clinical trials in this area. Increased awareness among the clinicians and randomized control trials in management of paraquat poisoning will definitely help in saving many lives in the future.

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