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Deliberate Self Harm By Paraquat Poisoning.

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ABSTRACT

Paraquat (1,1′-dimethyl-4,4′-bipyridylium dichloride) is a herbicide used to prepare land or control weeds in major food crops. Although banned in many countries, including the entire EU, But in a number of developing countries plantation workers and small farmers spray Paraquat to kill weeds. They are highly effective pesticides, but have been reportedly used for deliberate self harm in many parts of the world including India. As a result, several ten thousand people are poisoned every year and become ill. Thousands die a painful accidental death or commit suicide. There is no specific antidote available for Paraquat poisoning. It is important to establish the diagnosis early and to pursue aggressive decontamination and prevention of further absorption. However, high mortality rate is associated with Paraquat poisoning in spite of advances in treatment and supportive care. If paraquat was banned worldwide and so no longer available, many thousands of lives would be saved, whether from occupational poisoning, suicide, or accidents to children. We describe two cases of paraquat poisoning who developed serious toxicity following its ingestion and died of respiratory failure.

Keywords: Herbicide, Multi-organ failure, Acute respiratory distress syndrome, Acute kidney Injury

INTRODUCTION

Paraquat is one of the most widely used herbicides in the world. It is highly toxic to humans. It is easily and cheaply available in developing countries as they are in need of boosted food production and crop protection. It is produced commercially as a brownish concentrated liquid of the dichloride salt in 10 - 30% strength under the trade name of 'Gramoxone' and for horticultural use as brown granules called 'Weedol' at about 5% concentration(1). Paraquat poisoning has been widely reported worldwide, but only a few case reports are described in literature from India (2, 3).

Paraquat is the one of the most common pesticides causing death from suicide. They are highly effective pesticides, but have been reportedly used for DSH (DELIBERATE SELF-HARM) in many parts of the world including India (4). PARAQUAT alone is rated “highly toxic” (include arsenic or hydrogen cyanide). We describe two cases of Paraquat poisoning who developed serious toxicity following its ingestion and died of respiratory failure.

CASE REPORTS

CASE 1.

A 21yr old male came with alleged history of consumption of paraquat at his residence, initially he was treated at local hospital, and first aid treatment was given and referred to tertiary hospital for further management. On examination he was conscious, oriented with vomiting and corrosive injury of hard palate and pharynx, vitals were normal, lab investigations showed severe metabolic acidosis and lactic acidosis with increased total count and hypokalemia, provisionally he was diagnosed as paraquat poisoning as respiratory failure with severe metabolic acidosis. He was treated with stomach wash, shifted to MICU treated with IV fluids, and with supportive care, correction of hypokalemia, pantoprazole infusion. Patient was treated with N acetyl cysteine, methyl prednisolone and cyclophosphamide. Serial ABG was done which showed worsening of metabolic acidosis, HD catheter was inserted and initiated on dialysis. Gastroenterology opinion was sought and upper GI endoscopy was done which revealed extensive corrosive injury of lower esophagus, stomach and 1^{st} and 2^{nd} part of duodenum, conservative management was given with continued pantaprazole infusion. Patient had persistent metabolic acidosis and lactic acidosis. Patient became drowsy and respiratory distress, desaturated to spo2 80% on room air. Patient was intubated and connected to ventilator. Later patient had hypotension, initially resuscitated with IV fluids, started on vasopressor support. Patient was dialysed for second time. After two days patient deteriorated further requiring maximum supports of vasopressor and high flow oxygen requirement. Patient had cardiac arrest and resuscitation was done, but couldn’t be revived and declared dead. During postmortem examination corrosive injury was found over inner aspects of lips, hard palate, esophagus, stomach and duodenum. Lungs showed features of pulmonary edema with pleural effusion, liver showed fatty changes. However, blood and viscera was preserved for chemical analysis report.

CASE 2.

A 32yr female with alleged consumption of paraquat in her house, was shifted to hospital was treated conservatively, who was diagnosed as multi organ failure with shock, acute kidney injury associated with metabolic acidosis who died after two days. During postmortem examination corrosive injury of stomach was present. lungs were edematous with pleural effusion. Blood and viscera was preserved for chemical analysis.

These cases are reported to highlight the high mortality rate associated with Paraquat poisoning inspite of advances in treatment and supportive care.

DISCUSSION

Paraquat is considered the most poisonous herbicide in wide use. Paraquat is freely available in the Indian market for agricultural use. Paraquat is sold in about 130 countries for use on large and

small farms, plantations and estates and in non-agricultural weed control. The most frequent routes of exposure to Paraquat either accidentally or intentionally, in humans and animals are following ingestion or through direct skin contact(5). Most intentional self-poisoning occurs via ingestion, although injection with fatal consequence has been reported in Korea (6).

Paraquat causes extensive damage to the mitochondria of cells through the production of free radicals and oxidative stress, resulting in the interruption of important biochemical processes, causing cell death, and multi-organ failure (7). It distributes to all the organs.

Poisoning with Paraquat leads to both local and systemic effects. When consumed orally it results in corrosive injury to pharynx, stomach and duodenum as it is caustic in nature after absorption leads to hepatotoxicity, nephrotoxicity besides pulmonary fibrosis. Absorbed paraquat is sequestered in the lungs and causes release of hydrogen and superoxide anions which cause lipid damage in the cell membranes causing pulmonary edema or acute respiratory distress syndrome (ARDS)(8). Studies have identified it causes acute lung injury and pulmonary hypertension; acute kidney injury, leucocytosis; metabolic acidosis; increased levels of blood amylase, glucose, and creatinine, (9).

The percentage of survival is almost nil which means that once ingested the person will definitely die in a matter of days mostly from kidney failure, liver failure, heart failure, collapse of the lungs - resulting in ultimate death.

Acute paraquat self-poisoning is a significant problem in parts of Asia, the Pacific and the Caribbean (10). paraquat is a widely used suicide agent in third-world countries is due to its widespread availability, low toxic dose (10 mL or 2 teaspoons is enough to kill) and relative low cost. Paraquat is the one of the most common pesticides causing death intentional self-poisoning, i.e. suicide. There is concern in developing countries that the easy availability of pesticides leads to suicides which might not otherwise occur. It has a 60-70% mortality rate (11), much higher than many other agents. Prognosis in Paraquat poisoning is largely dependent on the amount of Paraquat absorbed. Mortality remains high even with prompt management and in spite of advances in treatment and supportive care (10).

Most cases are self-poisoning, but not all intend to die (12). This is very important, given that in countries such as developing nations intentional self-poisoning is “often a result of impulsive behaviour rather than the result of long-standing psychiatric problems”. Sudden anger and grief are common triggers (13).

Experience has shown that restrictions on availability of paraquat and other highly toxic pesticides have reduced deaths from poisoning (14). If Paraquat was not available the survival rate from attempted suicide would be significantly higher (15).

CONCLUSION

Paraquat is the most highly acutely toxic herbicide to be marketed over the last 60 years. Yet it is one of the most widely used herbicides in the world, and in most countries where it is registered it can be used without restriction. It is used on more than 100 crops in about 100 countries. There are numerous designs, management, mechanical and cultivational practices, as well as some plant extracts, that can be used instead of Paraquat, depending on the weed species and the situation. By far the biggest cause of non-occupational poisonings is intentional self-poisoning, i.e. suicide. This problem is central to the problem of Paraquat: if Paraquat were banned worldwide and so no

longer available, many thousands of lives would be saved, whether from occupational poisoning, suicide, or accidents to children. All other herbicides on the market have lower acute toxicity than Paraquat. There are some herbicides derived from natural plant extracts, such as pine oil or coconut oil, and these appear to have no or minimal health effects.

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