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PARAQUAT- A DEADLY POISON :REPORT OF A CASE AND REVIEW

General Medicine

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ABSTRACT

Paraquat is one of the most dangerous poisons in the medical field as Ingestion of small quantities (>10mL) may damage lung irreversibly and also lead to renal failure causing to death. The paraquat is sold in more than 130 countries around the world to be used in small as well as large farms. The easy availability of this poison makes it a commonly employed agent for suicide. More than 93% of deaths due to paraquat poisoning are suicidal in nature and most of them are occurring in developing countries. The clinical features vary from case to case based on the amount and type of compound consumed. As it is a fatal condition, early diagnosis and proper management is warranted which can reduce the morbidity and mortalities. But sometimes early diagnosis may not save the victim as in our case. We present a case wherein the victim succumbed to acute respiratory distress following consumption of paraquat.

KEYWORDS

Paraquat , Suicide , Respiratory Failure

INTRODUCTION

Paraquat (PQ, 1,1′-dimethyl-4,4′-bipyridinium dichloride), which was synthesized as a redox indicator in 1882, has been widely used as a herbicide since 1962. Suicides due to paraquat (PQ) are an important cause of morbidity and mortality, especially due to the absence of specific antidote. Consumption of PQ > 40 mg/kg causes acute multiorgan failure with death within the first 2 days, while < 20 mg/kg of PQ causes mild symptoms and most survive. Paraquat of 20–40 mg/kg causes severe mucosal damage followed by multiorgan failure. The few, who survive, die within 2–4 weeks due to lung fibrosis. The pulmonary alveolar cells selectively accumulate polyamines that are required for cellular functions. Being structurally similar to polyamines, PQ gets accumulated in these cells, causing selective delayed lung injury. Although there is no specific antidote, most patients receive steroids/antioxidants to reduce free radical damage. Severe paraquat poisoning is characterized by multiple-organ failure, involving mainly the lungs, kidneys, and liver. The lung is a major target organ in paraquat poisoning, and respiratory failure from lung injury is the most common cause of death. It is important to make an early diagnosis and to start appropriate treatment as soon as possible. This article presents a case of fulminant paraquat poisoning.

CASE REPORT

A 38-year-old female , weighing ~60kg was admitted to the emergency room with an alleged history of attempted suicide with consumption of >100 ml Paraquat (25% weight / volume) at 9 :00 am on 5/4/2022. Patient had experienced burning sensation around the lips ,nausea,vomiting and abdominal discomfort immediately following Paraquat ingestion . She was brought to the hospital 6 hours after the congestion of poison . Under immediate care at the hospital a gastric lavage with activated charcoal (1 gm/kg) was given ,urine samples for paraquat were sent and a ryles tubes was inserted predicting a difficulty in swallowing later on . Upon detailed enquiry with the relatives, there was alleged history of suicide of about 5 times in the past 1 year. Patient was a known case of Major Depressive Disorder and was on treatment with atypical antipsychotics. At the time of examination, patient was conscious and oriented. There were multiple self-inflicted cut wounds over both the wrists. Examination of the oral cavity showed mucosal erosion of tongue, lips and soft palate. On clinical examination, pulse rate was 88 beats per minute, regular, blood pressure was 110/80 mm hg, with a respiratory rate of 22 per min. Cardiovascular system was normal. There was breathing difficulty, but no added sounds on examination of the respiratory system. A computed chest tomography was done which did not report any abnormal findings. Initial complete blood count, electrolyte, and liver function tests were within normal ranges. On day 3 of admission blood chemistries showed leucocytosis,neutrophilia, deranged prothrombin time , elevated D-dimer levels , significantly elevated serum creatinine levels , deranged SGOT/ SGPT levels and total hyperbilirubinemia . Her renal function deteriorated, with markedly increased blood urea nitrogen (68 mg/dl) and creatinine levels (8.12 mg/dl), and her urine output decreased. Hemodialysis was started. Blood and urine cultures were sterile.

Urine examination revealed significant proteinuria and haematuria. ultrasound of the abdomen revealed enlarged liver with bright echotexture and increased echogenicity of bilateral kidneys .

Two- dimensional echo and electrocardiogram were normal . An upper gastrointestinal scopy was deferred since she was considered a high risk candidate for oesophageal rupture.Her arterial blood gas values on day 3 were pH 7.47, PaCO2 30 mm Hg, and PaO2 53 mm Hg. She required increasing supplemental oxygen to keep her oxygen saturation above 90%.In view of persistent hypoxia , she was intubated and put on mechanical ventilator on the 5 th day.

In the intensive care unit , she received IV fluids , intravenous methylprednisolone 1 g in 500 cc normal saline for 3 days, shifted to intravenous dexamethasone 4 mg 6 hourly.Antioxidant therapy was given in the form of intravenous vitamin C 1 g TID , intravenous aqueous vitamin K 1 g OD for 5 days and vitamin E (400 i.u /tab) 2 tabs QID. Detoxifying agents were given as , intravenous N-Acetylcysteine 150 mg /kg in dextrose solution, 500 ml, infused over 3 hours, subsequently 300 mg /kg in 5 % dextrose solution , 500 ml , continuous infusion at 20 ml/hr and Intravenous Glutaspa 600 mg BD.The patient did not respond to treatment, progressive multiple-organ failure ensued, and expired on the 10 th day post consumption of Paraquat as a result of respiratory failure.

Table 1: Investigations

day	CR	INR	SGOT	SGPT	pH	HCO3	pCO2
1	0.82	0.96	15	12	7.36	22	38
3	3.56	1.06	328	216	7.22	12	25
5	6.06	1.59	369	238	7.41	25	43
7	4.02	1.1	193	156	7.42	26	45
10	5.52	1.36	106	181	7.15	20	70

DISCUSSION

Paraquat poisoning is a relatively understudied and under-researched condition as evident in the search of literature. This is saddening in view of the knowledge that paraquat toxicity causes fatality in a large number of cases despite treatment in a tertiary care setting(2). With an expected mortality of 55 % in clinical settings being reported in healthy males without significant co-morbities, it remains a serious

health issue throughout the world(3). The concentrations of Paraquat available in the market vary from 10-40%. However, the lethal dose is calculated as being 35mg/kg which translates to as low as 10-15 ml of a 20% solution of paraquat available commercially(4).

The symptoms of paraquat poisoning may be classified as mild, moderate or fulminant depending on the route of intake, dose and the time of presentation to the hospital. Being a caustic agent, the local signs of inflammation will be seen even with mild doses. Taken orally, it shows oral and esophageal erosions along with gastric mucosal damage. The main activity of paraquat occurs in the lungs. Upon reaching the lungs via the blood, it induces lung injury which is morphologically characterized by an early destructive phase, in which the alveolar type I and type II epithelial cells are damaged; and a second proliferative phase defined by alveolitis, pulmonary oedema and infiltration of inflammatory cells. The entire activity occurs as a result of superoxide radicals and other free radicals created due to the accumulation of paraquat in the lungs(5).

Other theories put forward also hypothesize that inflammatory mediators and immune activation also play a major role in the damage caused due to the toxin(6,7). The biotransformation of the toxin occurs primarily in the kidney and hence urine concentrations are often high enough, however it also has a detrimental effect in the form of rapidly inducing acute kidney injury, thus forming a vicious circle in which the only way to remove the toxin is effectively shut down(8).

As there is no specific clinically proven antidote for paraquat poisoning, supportive treatment is given to avoid free radical injury to lungs (vitamins C and E),with pulse therapy using steroids (methylprednisolone or dexamethasone) to prevent pulmonary fibrosis, elimination of paraquat from circulation (hemodialysis), and gastric decontamination.In contrast, the use of oxygen can enhance the toxicity of Paraquat by providing more electron acceptors and should be given in lower concentrations to the hypoxic patients. In spite of advances in medical care, prompt treatment, and supportive care, mortality is high (mainly due to multiorgan system and respiratory failure) in patients with paraquat poisoning.Although there have been isolated case reports of survivors (mainly due to the smallness of the dose or effective and early treatment), an ingestion of a high dose or severe paraquat poisoning has a poor prognosis. At present, there is no specific antidote to paraquat poisoning. Therefore, it is recommended that the crucial focus should be on preventive measures and in case of exposure, when it has been ingested, the institution of aggressive decontamination to prevent further absorption.

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