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Original Research Article

Paraquat Poisoning: A case report

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Abstract

Paraquat (N, N′-dimethyl-4, 4′-bipyridinium dichloride), a brown syrupy liquid is an effective herbicide that has low chronic toxicity because of its rapid deactivation on contact with soil. A high dose of paraquat or severe poisoning has a poor prognosis. At present, there is no specific antidote to paraquat poisoning, hence the need to focus on prevention and in case of exposure or ingestion, aggressive decontamination to prevent further absorption. The prognosis is uniformly poor worldwide, including for those who treat aggressively with multimodal therapies. Long-term survivors are few, and have GI and pulmonary complications. Hence, prevention needs to be the utmost priority, and on exposure, aggressive decontamination should be initiated. Although it is a very common herbicide, there are very few cases reported from India, and awareness among people needs to be widened. Here is a case report of a 50 years old gentleman presented to the hospital with an alleged history of consumption of paraquat Poison by himself.

Keywords: Paraquat, Hemodialysis, herbicide, unconsciousness

Introduction

N, N′-dimethyl-4,4′-bipyridinium dichloride (paraquat), a pungent smelling corrosive liquid is currently the second highest selling herbicide worldwide [1]. Its accidental or deliberate ingestion is associated with a high mortality rate, and it produces both local and systemic toxicity [2]. PQ is an important herbicide used in agriculture; however, thousands of individuals succumb due to PQ intoxication every year in the developing world. PQ is a highly toxic compound and the fatality rate of PQ ranges between 60 and 80% [3] due to the lack of a specific antidote.

A PQ dose of 30 mg/kg may be fatal, which is equivalent to 8-10 ml of the 20% solution sold commercially [4]. PQ has been shown to cause significant damage to organs, including the lung, liver, kidneys and myocardium, with the highest concentration of PQ found in the lungs [5]. The prognosis of patients with multiple organ dysfunction syndrome (MODS) caused by fulminant poisoning (>40 mg PQ ion per kg of body weight) is extremely dangerous and patients may succumb within hours to a few days following ingestion [6, 7].

Its herbicidal properties were discovered in 1950s and first marketed in 1962. Once, it was encouraged by US for usage in Mexico to abolish marijuana plants. Presently, it is the second highest-selling weed killer globally and is available in a 20% solution form and that needs to be diluted before agricultural use [8]. Usually, adult cases of intoxication are due to suicidal attempts instead of homicidal or accidental exposure. The main acute systemic effects are pulmonary edema, convulsions, cardiac, renal and hepatic failure [9].

Deliberate ingestion of Paraquat leads to Jaundice, Mouth ulcer, Crepitation over Lung

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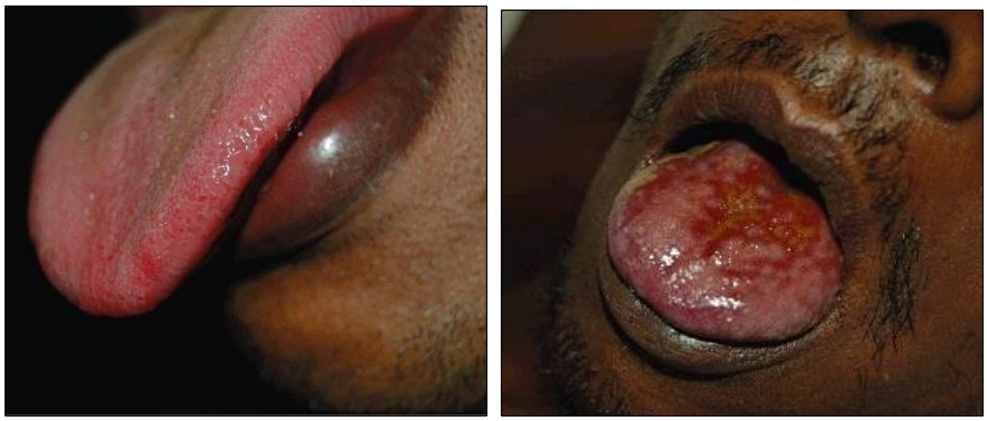
auscultatory areas, Hypoxia, Decreased Urine Output, pain in Abdomen. Paraquat gets concentrated inside cells, then it takes part in Redox cycling which consumes NADPH, which is one of the important antioxident, this redox cycling produces superoxide radicle which is highly Reactive Oxygen Species and ultimately Cellular Damage takes place. Furthermore, strategies for the management of PQ poisoning have focused on the modification of the toxicokinetics of the poison by either decreasing its absorption or enhancing its elimination [10]. Here we are reporting a case of 50 years old male, who was presented to us after 7 days of Paraquat ingestion by himself.

Case report

A 50 years old gentleman presented to our hospital who was a resident of Kanpur Nagar with an alleged history of consumption of paraquat Poison by himself [Suicidal Attempt], 7 days prior to admission (11 May). Four hours after consumption of the poison, he was found by his relatives unconscious and was taken to a local hospital where he was given gastric lavage, activated charcoal, and managed conservatively with intravenous fluids, antiemetics, and H2 blockers. At this point, he was referred to our hospital for further management. Patient came to our hospital on 18 may at 05:40 pm. The patient presented with shortness of breath, abdominal pain in Right Hypochondriac region, yellowish discoloration of sclera, nausea & vomiting, generalize body weakness. Patient also had a history of alcohol intake for past 15 years, 2 quarters of desi liquor a day. Patient was already admitted in some local nursing home in Kanpur, before admission here in our Hospital.

On presentation, Blood pressure was 150/90 mm of Hg, SpO2 was 94% on Room air. Bilateral fine crepts were present over infrascapular region. At the time of arrival at our center, his main complaints were a progressive difficulty in swallowing, associated with multiple painful oral ulcers. He was admitted in the hospital for further evaluation and management. On admission, his SpO2 dropped to 80%, then he was provided with nasal mask with 5 liters of O2 & patient shifted to casualty. Still his SpO2 was not raising 84% on 5 liters of oxygen, then the patient was put on high flow nasal mask on 10 liters oxygen, then he started maintaining saturation 90%, whenever the mask was tried to remove, his saturation used to drop below 80%.



Picture 1 & 2: Oral and tongue lesions

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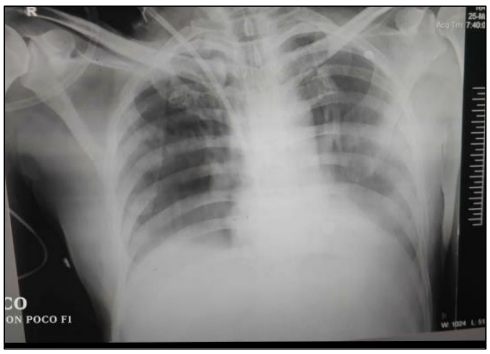

Fig 3: Chest X-ray

On presentation, the readings were:

Haemoglobin	13.1 gm/dl
TLC	8300 cells/µl
Platelets	77,000 per microliter
Total bilirubin	8.2 mg/dl
Direct bilirubin	3.9 mg/dl
Serum albumin	3.0 g/dl
SGOT	144 U/l
SGPT	133 U/l
Serum creatinine	9.0 mg/dl
Serum blood urea	24 mg/dl
Serum uric acid	11.5 mg/dl
Serum sodium levels	138 mmol/l
Serum potassium levels	3.0 mmol/l
PT	17.6 sec
INR	1.3
Viral markers (HCV, HIV, HBsAg)	Negative
Blood group	B Positive

On evaluation, serum uric acid levels, serum creatinine, SGOT, SGPT levels, total and direct bilirubin levels were highly increased. Potassium levels, serum albumin levels were reduced and platelets were decreased than normal range; while Blood urea and serum sodium levels were normal. Ryles tube, Foleys Insertion was done at the time of admission. Then after Blood investigations, central line was put & 1st hemodialysis was done on 21st May. Similarly, 4 sessions of Hemodialysis were done. . He was initiated on a liquid diet and a gastroenterology opinion was sought for the management of possible esophageal strictures.

	On presentation	**After 4 sessions of hemodialysis**
Serum creatinine	9.0 mg/dl	3.0 mg/dl
Blood urea	24 mg/dl	118 mg/dl
Serum uric acid	11.5 mg/dl	1.2 mg/dl
Serum sodium levels	138 mmol/l	136 mmol/l
Serum potassium levels	3.0 mmol/l	3.3 mmol/l

Then the patient was shifted to the general ward, he was still maintaining saturation 90% on high flow nasal mask. His chest X-ray was normal. He was discharged in a stable condition and was advised to be on regular follow-up with serial chest x-rays and spirometry to rule out pulmonary fibrosis.

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Discussion

Poisoning by pesticides is a major problem in low-income and middle-income countries. A systematic review of data extracted from 2006 to 2015 concluded that pesticides were responsible for almost 20% of global suicides leading to approximately 150 000 fatalities per year over the afore-mentioned period [11]. Unfortunately, this is a less commonly researched topic. Although organophosphate accounts for the majority of the hospital admissions, poisoning by paraquat compounds is a major medical problem as it is associated with a very high mortality (case fatality rate of 50%-70%) and morbidity [12]. Paraquat is a toxic bipiridyl compound, and it is lethal even in very small amounts (15-20 mL of 20% w/v). The mode of poisoning is usually suicidal or accidental; and it should always be suspected in cases of corrosive oral and oesophageal ulcerations along with features of hepatic, renal or pulmonary involvement [13].

Since bipyridyl salts are caustic, the gastrointestinal tract can be severely injured after ingestion of a concentrated solution. Once large concentration of this poison accumulates in lungs or renal cells, it leads to generation of toxic reactive oxygen species through redox cycling, which devastate cellular defensive system. Renal failure can result due to the direct toxicity and hemodynamic changes. Conservation of renal function is vital to reduce plasma paraquat levels and thereby reduce accumulation in lung cells [14].

Paraquat is incompletely absorbed from the gastrointestinal tract and mainly exerts its toxicity through production of reactive oxygen species (ROS) which causes cell damage by several mechanisms, such as mitochondrial toxicity, lipid peroxidation, oxidation of nicotinamide adenine dinucleotide phosphate (NADPH), activation of nuclear factor kappa B and apoptosis [15]. It targets both type I and II pneumocytes in the lung, leading to acute alveolitis. The initial 'destructive alveolitis' is followed by a 'proliferative' or 'cellular' phase, which ultimately leads to lung fibrosis [16, 17]. Hepatorenal involvement in the form of acute tubular injury and hepatocelluar damage is also common. Mucosal ulceration in the oral cavity and tongue, also known as 'paraquat tongue' is seen within the first few days of poisoning [17].

Intoxication of parquat ingestion can be categorized to mild, moderate, and fulminant. Mild intoxication can happen with doses ≤20 mg/kg, which usually produce minor gastrointestinal problems like transient vomiting, diarrhea, and oropharyngeal burns, but usually complete recovery is possible. Moderate intoxication can occur with doses between >20 mg/kg and < 50 mg/kg of the poison. Patient may suffer lung injury, pulmonary fibrosis, acute renal failure and in majority of cases, death occurs within 2-3 weeks. Fulminant intoxication of ≥50 mg/kg of the poison, may lead to death within 3 days, because of multiple organ failure. In patients who survive longer, fibrotic changes in the alveoli result in gas exchange interference in the lungs and may progress to ARDS [10, 15].

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) [18-21], with pulse therapy using steroids (methylprednisolone or dexamethasone) and cyclophosphamide to prevent pulmonary fibrosis [22, 23], elimination of paraquat from circulation (hemodialysis), and gastric decontamination [21, 24-26]. 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 [24, 27, 28]. 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 [19, 21, 29-33]. Although there have been isolated case reports of survivors (mainly due to the smallness of the dose or effective and early treatment) [33], an ingestion of a high dose or severe paraquat poisoning has a poor prognosis.

Conclusion

In the present case report, a total of 4 sessions of hemodialysis were done. Fine crepts were persistent. Patient was shifted to ICU, intubation was done, even on FiO2-100%, patient was able to maintain saturation 75% only and patient further deteriorates. 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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