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Case Report

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A Case Report of Paraquat Poisoning with Acute Kidney Injury Managed Conservatively without Long-term Pulmonary Sequale

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Abstract

Paraquat's chemical name is N, N′-dimethyl-4, 4′-bipyridinium dichloride. Due of its quick inactivation upon contact with soil Paraquat has minimal chronic toxicity. In India, instances of this pesticide toxicity are rare despite its broad availability. Ingestion of paraquat can cause severe and frequently lethal poisoning. There is currently no known treatment for paraquat poisoning, thus efforts should be directed on prevention and, in the event of exposure or ingestion, rigorous decontamination to stop further absorption. The prognosis is dismal everywhere, even for individuals who get rigorous multimodal therapy. The lungs, GI (gastrointestinal) tract, kidney, liver, heart, and other organs can all suffer life-threatening consequences from ingesting toxic levels of paraquat, which can be deadly. There aren't many long-term survivors, and they often experience lung and GI issues. The paraquat lethal dosage is so low that >10 ml of the toxin can irreversibly harm the lungs. Without a solid history, a particular clinical symptom, or a diagnostic test, diagnosis is frequently challenging. One of the most significant methods of poisoning is exposure by inhalation. Our patient was found to have consumed a product that included paraquat in excess of the deadly amount, and despite our best efforts, we were unable to rescue her. This indicates the high fatality rate of this type of poisoning. Adults are twice more likely to be poisoned by paraquat during suicide attempts than through accidental exposure. Pneumomediastonium is a consequence of paraquat poisoning that affects 20% of patients and has a 100% fatality rate.

Keyword: Paraquat poisoning, Mortality, Acute renal failure, Pulmonary fibrosis, Herbicides

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Introduction

Pesticide and other agricultural chemical poisoning is a significant global public health issue, particularly in poor nations. The second most used herbicide in the world right now is N, N′-dimethyl-4, 4′-bipyridinium dichloride (paraquat). It is a green, poisonous, caustic liquid with a strong odor [1, 2]. Due of its quick inactivation upon contact with soil it has minimal chronic toxicity [3, 4]. Its herbicidal qualities were initially commercialized in 1962 after being discovered in the 1950s. It is available as a 20% solution that must be diluted before being used in agriculture. It creates both local and systemic toxicity and has a high fatality rate when accidentally or purposefully consumed. Despite its widespread availability, poisoning with this pesticide is not a typical occurrence in India. Humans have an LD50 of around 35 mg/kg, which equates to about 10-15 ml of a 20% solution. Adult paraquat poisoning for suicide attempts is twice more frequent than poisoning from accidental exposure. Paraquat enters the lungs, kidneys, liver, and muscles after intake since these organs absorb a lot of blood. Lung is a significant target of paraquat poisoning and respiratory failure occurs with lung damage since the concentration in the lung parenchyma is roughly 10–20 times greater than that in plasma due to its active absorption by type 1 and 2 pneumocytes. Lung fibrosis brought on by lipid peroxidation is one of the major signs of paraquat toxicity. Pulmonary edema, convulsions, cardiac, renal, and hepatic failure are the primary immediate systemic consequences. According to earlier research, pneumomediastinum is a complication of paraquat poisoning that affects around 20% of patients and has a death rate of about 100%. We shall discuss a case of paraquat poisoning for a suicidal attempt in the article that follows.

Case Report

A 16-year-old female was brought to emergency room with alleged history of consumption of paraquat poison 24 % SL (Ruler contact herbicide) approx volume of 2-4 ml at her residence.

The patient complained of pain in abdomen which was associated with 2-3 episodes of vomiting. The patient was taken to an outside hospital where the patient was initially managed with gastric lavage and brought to our hospital. There was no history of decreased urine output, loose stools, seizures. At the time of examination, the patient was conscious, coherent, and cooperative. On examination of the oral cavity no mucosal erosion of tongue, palate, and lips with oral bleeding

was seen. On clinical examination patient had stable vitals. The cardiovascular system was normal. There was no difficulty in breathing. Pupils were bilaterally equal and reactive to light. The chest X-ray was normal. She had high serum urea (51 mg/dl) and creatinine (4 mg/dl) on the day of admission that gradually reduced to normal without the need for dialysis. Patient was slightly anaemic throughout the course of admission. Blood and urine cultures were sterile. No derangement of liver function test was documented. The urine examination was normal. There was mild increase in echotexture of b/l kidneys on USG abdomen. The electrocardiogram was normal. Upper GI endoscopy showed normal mucosal study.

The patient was treated with Injection N- Acetyl Cysteine 1.2 g thrice daily for one week, Inj Hydrocort 100 mg was given TID. Along with the given above treatment, the patient was put under antibiotic coverage with Inj Monocef 1 gm iv bd for the hospital stay. Inj Pan 40 mg iv OD was given for one week. Patient was put on NBM till upper GI endoscopy was done, and later patient was allowed on oral diet as the upper GI endoscopy report was normal. Patient was given maintenance IV fluids during hospital stay. The serum creatinine levels normalised over the next one week without any need for haemodialysis. The patient was discharged in a stable condition after one week and was asked to follow up to look for any delayed complications like pulmonary fibrosis.

Discussion

Paraquat (N, N'-dimethyl-4, 4′-dipyridylium) is a broad-spectrum liquid herbicide. Commercial paraquat formulations are typically offered for sale as liquid concentrates with a 20% to 42% w/w concentration range. These solutions are accessible as water-soluble dichloride salts for use in agriculture. There are products on the market that contain paraquat in addition to being offered as a single active component and in mixtures with other herbicides like sodium chlorate and 2,4-dimethylamine.

The bipyridyl substance paraquat damages cells directly by producing superoxide and other reactive oxygen species as well as nitrite radicals, which damage cell membrane lipids and lead to oxidative free radical damage [5, 6]. After being ingested, paraquat is trapped in the lungs and can cause hepato/nephrotoxicity, and pulmonary fibrosis.

The most prevalent symptom in a 17-patient Indian series was vomiting (100% of the time), which was followed by altered sensorium (59%), oral ulceration or dysphagia (53%), dyspnea (41%), and loose stools (24%) [7]. Both local and systemic toxicity are caused by the clinical course, which is often dosage dependant. Ingestion of paraquat causes corrosive harm to the gastrointestinal system as well as inflammation of the tongue, oral mucosa, and throat, as well as renal tubular necrosis, hepatic necrosis, and pulmonary fibrosis. The hallmark of paraquat poisoning is lung involvement in the form of widespread alveolitis and eventual pulmonary fibrosis. The pulmonary effects of paraquat poisoning start with widespread consolidation and progress over a few days to cystic lesions, localized fibrotic lesions, and extremely lethal fibrotic lesions. It is believed that ingesting huge doses will always result in cardiogenic shock and multiorgan failure.

With a dosage of less than 20 mg/dl, can result in erosions of the tongue, oral mucosa, and corrosive harm to the GI tract. With moderate toxicity, ingestion of 20–50 mg/dl can cause lung fibrosis, hepatic necrosis, and renal tubular necrosis; death often occurs within two to three weeks. Death from multiorgan failure and shock occurs in fulminant poisoning (consumption of more than 50 mg/dl), and it typically takes 3 days. Histopathological findings in fatal paraquat poisoning instances range from lung congestion, edema, and bleeding to severe pulmonary fibrosis.

To confirm the diagnosis, a urinary dithionate test for paraquat identification is also utilised. The likelihood of survival is good if the urine paraquat content is less than 1 mg/L within 24 hours following paraquat poisoning. 12 hours after admission, plasma values exceeding 1.6 pg/ml were shown to be fatal [8]. Nasogastric tube fixation, gastric lavage with regular saline, gastric lavage with charcoal-sorbitol powder, forced alkalinized diuresis, and haemodialysis are typical therapies which are employed in treating paraquat toxicity. Hemoperfusion with activated charcoal is helpful for up to 4 hours following paraquat intoxication. Salicylates, vitamin E, vitamin C, and N-acetylcysteine are examples of antioxidants that can be helpful in preventing the production of free radicals, which can cause inflammation, and nuclear factor kappa B. Since there is no specific, clinically validated treatment for paraquat poisoning, supportive measures are taken to prevent lung damage from free radicals, to prevent pulmonary fibrosis with pulse therapy using steroids (methylprednisolone or dexamethasone), to remove paraquat

Figure 1: https://www.novaagrisciences.com/products/RULER.php

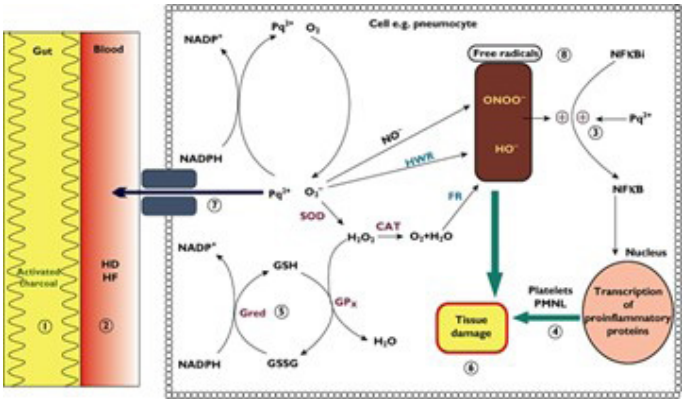

Figure 2: Illustration of the effects of paraquat poisoning inside a pneumocyte and prospective treatment locations [14]. SOD, superoxide dismutase; CAT, catalase; Gred, glutathione reductase; Gpx, glutathione peroxidase; FR, Fenton reaction; HWR, Haber-Weiss reaction. 1–8: potential sites of action by available treatment options. 1: activated charcoal and Fuller's earth; 2: dialysis; 3, 4, 6 and 8: salicylates; 5 and 8: N-acetylcysteine; 7 (P-glycoprotein induction): dexamethasone; 4: immunosuppression.

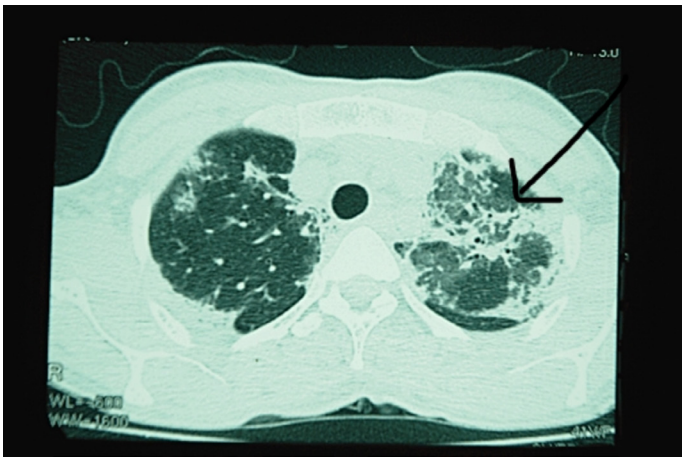

Figure 3: High resolution CT imaging of the chest 11 days after paraquat exposure, showing bilateral pulmonary fibrosis [14]. (Arrow indictating pulmonary fibrosis)

from the bloodstream (haemodialysis), and to decontaminate the stomach. In cases of mild poisoning, paraquat is primarily eliminated by the kidneys within 24 hours. The terminal elimination half-life, however, may be more than 100 hours. To stop the poison from being absorbed, gastric lavage and adsorbents such activated charcoal (1-2 g/kg) and Fuller's earth (1-2 g/kg) should be started as soon as feasible [9]. If administered within 4 hours of intake, hemoperfusion has been proven to be helpful in lowering the paraquat level [2]. Only as a supportive therapy for individuals who experience acute tubular necrosis is haemodialysis employed. Immunosuppression's function is currently being researched [10]. According to a recent Cochrane meta-analysis, patients who got standard care together with glucocorticoids and cyclophosphamide had a reduced probability of passing away at the end of their treatment than those who just received standard care [11]. In contrast, oxygen should be administered to hypoxic patients in lower amounts since it might increase the toxicity of paraquat by giving more electron acceptors [12, 13].

Good prognostic indicators were young age, percutaneous or inhalational route, exposure to less paraquat, and lower levels of leucocytosis, acidosis, renal, hepatic, and pancreatic failures upon admission. Renal failure, oesophageal erosions, esophagitis, and strictures were the frequently occurring late sequelae among survivors Another significant late consequence of paraquat poisoning is progressive pulmonary fibrosis, which can result in mortality 2–3 weeks later from hypoxia and respiratory failure. Despite improvements in medical care, quick intervention, and supportive care, paraquat poisoning patients have a significant fatality rate (mostly because of multiorgan failure and respiratory failure). A large dosage of paraquat or severe paraquat poisoning has a bad prognosis, although sporadic case reports of survivors (mostly owing to the modest amount or successful and prompt treatment) are being reported. Therefore, it is advised that the essential emphasis should be on preventative measures and, in case of exposure, the introduction of vigorous cleaning to stop further absorption, once it has been consumed [14].

Conclusion

Paraquat is one of the commonly used herbicides which has very high mortality. This toxin has no known particular antidote. The cornerstone of therapy is early diagnosis, rapid history collection, vigorous decontamination using stomach adsorbents. Oropharyngeal ulcerations, oesophageal erosions, renal failure, and pulmonary fibrosis are among the late effects of paraquat use. For the purpose of determining the severity of the intoxication and forecasting the likelihood of survival, serum and urine concentrations of samples taken at known intervals after intake must be established. The therapeutic procedures of hemoperfusion and dialysis are advised if the patient arrives early in order to prevent pulmonary and multi-organ failure.

Acknowledgements

None.

Conflict of Interest

None.

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