Case Report

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Paraquat Poisoning and AKI – A Rare Pediatric Case Report in a Tertiary Care Hospital

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Abstract

Paraquat is a widely used synthetic herbicide in agriculture. Ingestion of toxic doses of paraquat can be fatal with life threatening effects on the kidneys, lungs, gastrointestinal tract (GIT), liver, heart, and other organs. There is no specific antidote or effective life-saving treatment for paraquat poisoning and its prognosis is very poor worldwide. Here we report a case that presented with acute renal failure, shock, GIT bleeding with oral mucosal ulceration, and pulmonary involvement due to accidental paraquat poisoning and delayed hospitalization after poison ingestion. In spite of partial improvement, the patient expired after two weeks of admission. Therefore, any case of paraquat poisoning must be hospitalized and, if needed, referred to equipped centers as early as possible after poison ingestion.

Keywords: Paraquat; Acute Renal Failure; Herbicide; Pulmonary Fibrosis.

Conflict of interest: The authors declare no conflict of interest.
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Introduction

Paraquat (N, N′- dimethyl L-4,4′- bipyridium dichloride) is a widely used synthetic, nonselective, contact herbicide in the Asia-Pacific region (1). It is available in granule, liquid, and aerosol form and may be combined with diquat (2). The annual consumption of paraquat is increasing sharply in Bangladesh due to a government ban on WHO class-1 pesticides in 2000. There is no reported incidence rate in our country yet. The World Health Organization classifies paraquat as a 'class II moderately toxic pesticide'. Mild and moderate intoxication occurs with doses of ≤20 mg/kg and 20-50 mg/kg of the poison, respectively. In moderate intoxication, death may occur within 2 weeks. Fulminant intoxication occurs with ≥50 mg/kg of the poison. Ingestion of toxic doses of paraquat can be fatal with life-threatening effects on the lungs, gastrointestinal tract, kidneys, liver, heart, and other organs. The high mortality rate of this herbicide ingestion is due to multi- organ failure and lack of a specific antidote. There is no effective management for this type of poisoning in humans (3,4). This case report of paraquat poisoning is presented to emphasize the pitfalls of the management with a simple review of related articles.

Case report

A 10-year-old boy from Ramu, Cox's Bazar was admitted to Chittagong Medical College Hospital (CMCH) with complaints of sore throat, fever, respiratory distress, and oral ulceration for 4 days. Initially, the patient was treated locally for 4 days and was then referred to CMCH. On further enquiry, the boy said that he drank the leftover liquid in a Coca Cola bottle in a playground 4 days ago as he was thirsty. He was treated in the ENT Department followed by the Pediatric Department and then

transferred to PICU. On examination in the PICU, he was drowsy, disoriented, not pale or icteric, tachypneic, and tachycardic. His temperature was 100˚F, BP was 80/50 mmHg, SPo2 was 90% without oxygen, urine output history was confusing, and CBG was 8.9 mmol/L.

There was bright red excoriation of the oral mucosa, tongue and lips (Figure 1).

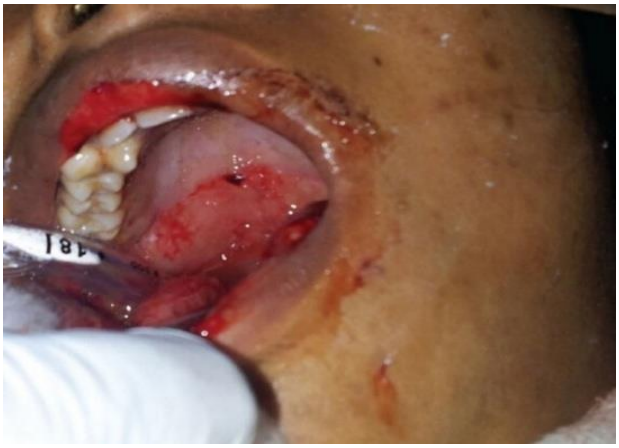

Figure 1. Extensive oral ulceration

The patient also passed black tarry stool. Other systemic examinations were normal. The initial complete blood count was also normal. Blood chemistry showed hypernatremia (S.Na–160.8 mmol/l) and hypokalemia (K–2.9 mmoL/L). Serum creatinine was 3.5mg/dL, SGPT was 428U/L, prothrombin time (PT) was 19 sec, and serum albumin was 2.3g/dL. Chest X-ray (Figure 2) and abdominal ultrasound revealed no abnormality. Shock was managed with adequate fluid therapy and electrolyte abnormality was corrected accordingly. The patient was given methylprednisolone injection, ceftriaxone injection, albumin 20% injection, N-acetylcysteine injection, vitamin K1 injection, fresh frozen plasma, vitamin C and E, oral lactulose solution, and ursodeoxycholic acid. Oral care was maintained by Gentian violet and miconazole 2% oral gel. With these treatments, a normal urine output was maintained and serum creatinine reduced to 1 mg/dl. On the 6th day of admission, the patient again developed shock. Shock was managed accordingly. His renal function improved but there was respiratory distress, cyanosis and persistent hepatic failure (SGPT: 428U/L, PT: 26.4 sec). In spite of all available treatment facilities, the patient developed respiratory failure and expired on the 12th day of admission.

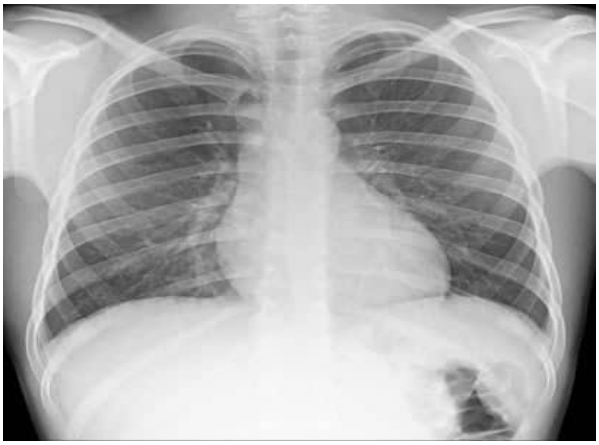

Figure 2. Chest X-ray on admission

Discussion

The clinical manifestations that follow paraquat ingestion depend upon the quantity ingested (5,6). Ingestion of small quantities usually leads to toxicity in the two key target organs (kidneys and lungs) over the next 2–6 days. Ingestion of large amounts of the liquid concentrate results in fulminant organ failure as renal and hepatic failure, severe lung injury or pulmonary edema, cardiac involvement, and convulsions due to CNS involvement. Mortality is still well over 50% in the fulminant form (7). The history of the ingested amount was confusing in our patient but multiorgan failure indicated at least moderate consumption of paraquat.

A universal feature of paraquat concentrate ingestion is gastrointestinal toxicity. Mucosal lesions of the mouth and the tongue (paraquat tongue) begin to appear within the first few days, which may become ulcerated with bleeding. Sandhu JS et al. reported an Indian series of 17 patients with paraquat poisoning in whom the most common symptom was vomiting (100%) followed by altered sensorium (59%), oral mucosal ulceration or dysphagia (53%), dyspnea (41%), and loose stool (24%) (8). In our patient, the initial chief complaints were sore throat, oral mucosal ulceration, and GIT

bleeding (melena). As there was mucosal excoriation and black tarry stool, it was assumed that the whole GIT was involved. Potassium loss through the intestines may be the cause of hypokalemia in this case. Elimination of paraquat is mainly by kidneys, usually within 24 hours after ingestion. The final elimination half-life can exceed 100 hours. In case of paraquat poisoning, there is a high probability of survival if its urinary concentration is < 1mg/ L within a day (1). Our patient presented with clinical features of acute renal failure with high a creatinine level. Here the cause of renal failure was multifactorial- maybe due to hypovolemia, circulatory failure or direct toxicity related to redox cycling. As features of renal failure improved by adequate fluid therapy, dialysis was not arranged for our patient.

Liver toxicity or liver failure is another common picture of paraquat poisoning like high levels of SGPT, which was present in our case. During admission, the patient was drowsy with impaired consciousness. Any impairment of consciousness usually indicates severe toxicity resulting from hypoxia, hypotension and severe acidosis (7). The major effect of large quantities of paraquat follows its accumulation in the lungs with lung cell damage causing decreased gas exchange and respiratory impairment. The pulmonary involvement has two phases: an acute alveolitis over 1–3 days followed by a secondary fibrosis. Most of the patients typically develop increasing signs of respiratory involvement over 3–7 days and ultimately die of severe anoxia due to rapidly progressive fibrosis even after 5 weeks (7,8). In our case, the initial chest X-ray was normal and the patient developed respiratory distress on day 11, so a chest CT scan could not be done to evaluate fibrosis.

Regarding treatment, removal of ingested paraquat by induced emesis or by gastric lavage with activated charcoal must be ensured immediately in a health care center. Activated charcoal should be given in repeated doses of 60 gm by gastric tube every two hours for three or four doses. Clay (Fuller's earth) is also an effective adsorbent. As the child was brought to the hospital from a rural area (after 4 days of ingestion), it is assumed that paraquat was absorbed by this time, which is why gastric lavage, activated charcoal, or Fuller's earth were not tried. Unfortunately, they were not tried in the local health care facility either. Supplemental oxygen should be withheld unless the pO_2 is below 70 mmHg because oxygen may contribute to the pulmonary damage mediated through lipid peroxidation (9). Hemoperfusion using activated charcoal has been shown to decrease the paraquat level, but there is insufficient data to support its survival benefit in humans (10). Hemodialysis is used as a support for acute renal failure, but it does not increase the clearance of the substance as it is rapidly distributed in the lungs and other organs (8). Immunosuppression with a combination of cyclophosphamide and methylprednisolone has been shown to be beneficial in moderate-to-severe cases by prevention of ongoing inflammation (11). Lin et al reported the therapeutic effect of high dose cyclophosphamide and glucocorticoid with a survival of about 75% (12). An intensive care unit study and a meta-analysis conducted by Agarwal et al concluded that immunosuppressive therapy with cyclophosphamide and glucocorticoids had a potential role in management of paraquat poisoning in moderate to severe poisoning cases (12,13).

Subsequent management includes antibiotics for supervening infection and supporting renal function with hemodialysis or filtration. Potent analgesics such as opiates may be required to alleviate intense pain from gastrointestinal tract injury, ulceration, and inflammation. Some antioxidants like vitamins C and E have been clinically used to protect against free-radical toxicity. N-acetylcysteine is also used as an antioxidant because of its free radical scavenging property; it also increases intracellular glutathione levels. Therefore, it will provide a protective effect for lung parenchymal cells (14). Since there is lack of clear evidence-based therapy for paraquat intoxication, different approaches have been tried for supportive management. In the present case, our therapeutic approach did not improve the patient's outcome.

The outcome depends on the severity of the poisoning and the time taken to avail medical help. The high mortality rates are due to the toxicity of the compound itself and lack of a specific antidote (15). Hypokalemia is a biochemical signal of poor prognosis for acute paraquat poisoning (16). Progressive pulmonary fibrosis is an important late

complication of paraquat toxicity that can cause death 2–3 weeks later due to hypoxia and respiratory failure (8). In our case, the cause of death after 16 day of paraquat ingestion could be lung fibrosis or respiratory failure.

We reported our experience of acute paraquat poisoning along with its clinical presentation and outcome. The data on pediatric paraquat poisoning in our country is very scarce. Therefore, any unexplained combination of gastrointestinal symptoms, acute renal injury, and respiratory failure of a rural pediatric patient should raise a suspicion of paraquat toxicity, even in the absence of ingestion history.

Conclusion

As the outcome of paraquat poisoning is usually fatal, all cases of paraquat intoxication regardless of symptoms must be hospitalized and, if needed, the patient should be referred to equipped centers as early as possible to receive the maximum available care for better management.

Conflict of Interest

The authors declare no conflicts of interest.

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