Case Series

A case series on survivors of paraquat poisoning complicated by acute kidney injury and acute lung injury

Debasis Mondal[1], Prantick Kumar Bhunia[2]

From [1]Junior Resident, [2]Assistant Professor, Department of General Medicine, Medical College, Kolkata, West Bengal, India

ABSTRACT

Paraquat poisoning is nearly fatal. Death occurs by respiratory involvement. It has no antidote and also treatment protocol is lacking. Here, we present a case series of two survivors of paraquat poison who survived after proper care. Both of them were young and ingested in small quantities. Both of them had acute kidney and lung injuries. Patient 2 had an additional liver injury. Here, we have gathered some already published documents on treatment and outcome with a brief review.

Key words: Paraquat, Paraquat poisoning, Paraquat survivor, Paraquat treatment

N, N′-dimethyl-4, 4′-bipyridinium dichloride, or Paraquat is a synthetic, non-selective, contact herbicide that acts by generating reactive oxygen species [1]. It is a redox-active molecule that undergoes two one-electron reductions in steps (methyl viologen). In humans, paraquat acts by generating free radicals and destroying the lipid cell membrane and organelles. Paraquat poisoning has a high mortality rate due to both its local and systemic effects [2]. On ingestion, it is absorbed from the stomach; approximately 20% of the ingested poison gets absorbed. It selectively gets concentrated into lung tissue (10–20 times that of plasma) due to high perfusion [3]. It causes diffuse alveolitis and consequently pulmonary fibrosis; this is pathognomonic of paraquat poisoning. Other organs which get affected are the kidney, liver, and heart. It mostly gets eliminated unchanged, actively through kidneys, within 12–24 h following ingestion. This leads to vacuolation in proximal convoluted tubules resulting in renal tubular necrosis. Liver injury occurs due to mitochondrial damage and endoplasmic reticulum degranulation. Clinical features include coated tongue with necrotic slough (paraquat tongue), pain, and dysphagia due to esophageal ulceration. Systemic manifestations include nausea, vomiting, epigastric pain, and consciousness is not generally impaired (unless hypoxemia sets in). Lung involvement can be diagnosed with a chest X-ray and acute respiratory distress syndrome develops with gradual lung injury. Rising urea and creatinine and decreasing urine output signify renal impairment. High urine output is desirable. Hepatic involvement is diagnosed by raised bilirubin, aspartate transaminase (AST), and alanine aminotransferase (ALT); acute liver failure is not seen. Despite early interventions, most of the unlucky patients die within 24 h with substantial poisoning and after 1–2 weeks with lower doses [4]. However, some patients do survive.

Here, we report a case series of two young individuals, who developed renal and respiratory complications but recovered completely. Assent of patients and informed consent from their parents has been taken to publish their cases as a series.

CASE SERIES

Case 1

A 15-year-old girl presented to us 28 h after self-ingestion of 20 mL of 24% paraquat, following a family dispute. Identification of the poison was based on recollection by the victim herself and examination of the bottle (Fig. 1). On examination, she had ulcers in the oral cavity, inner lip, and coated tongue. Other general and systemic examinations were unremarkable. Electrocardiogram showed no changes and chest X-ray was normal.

After admission, she became oliguric. Her initial urea and creatinine were 42 and 1.4, respectively; which increased to 117 and 6.5, respectively (after first session of hemodialysis). She was started on hemodialysis immediately after admission and received a total of seven sessions of hemodialysis (3/week) over 2 weeks. Upper gastrointestinal endoscopy was deferred. Initially, she was kept nil per mouth for 24 h following admission and received intravenous fluids. She was allowed clear oral liquids under supervision and gradually received semisolid and solid food. She

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Correspondence to: Debasis Mondal, No. 88, College Street, Kolkata - 700 073, West Bengal, India. E-mail: 2015dm5@gmail.com

developed a mild cough 2 days following admission, chest X-ray was unremarkable, was treated with intravenous ceftriaxone and ambroxol syrup. Her oxygen saturation by pulse oximetry was normal throughout.

She received intravenous N-acetylcysteine 1 g over 3 h and 1 g twice daily thereafter, from day 1 of admission. She also received vitamin E (400 IU) 2 tabs thrice daily. Her hemoglobin level dropped following hemodialysis which was managed with packed red cell transfusion. She showed no signs of hepatic and pancreatic injury both clinically and biochemically. Psychiatric evaluation and counseling were done during his stay. She recovered gradually. She was discharged on day 20 following admission with urea and creatinine values of 13 and 0.8, respectively. Chest X-ray showed that no infiltrates and electrocardiogram showed no evidence of cardiac involvement. 2 weeks following discharge her urea was 21, creatinine 0.9, AST 18 and ALT 21. 6 weeks following discharge her urea was 23, creatinine 0.9. Chest X-ray and electrocardiogram remained unremarkable.

Case 2

A 16-year-old boy presented to us 2 h after self-ingestion of 10 mL of 24% paraquat, after getting scolded in school. He got this poison from a shopkeeper in a plastic pouch. On examination, he only had an ulcer on his tongue. Other general and systemic examinations were unremarkable. The electrocardiogram and chest X-ray were normal.

After admission, he received one hemodialysis immediately. His initial urea and creatinine were 95 and 2.7 respectively; which increased to 136 and 3.5, respectively (after hemodialysis). He received no further hemodialysis, as his family withdrew consent.

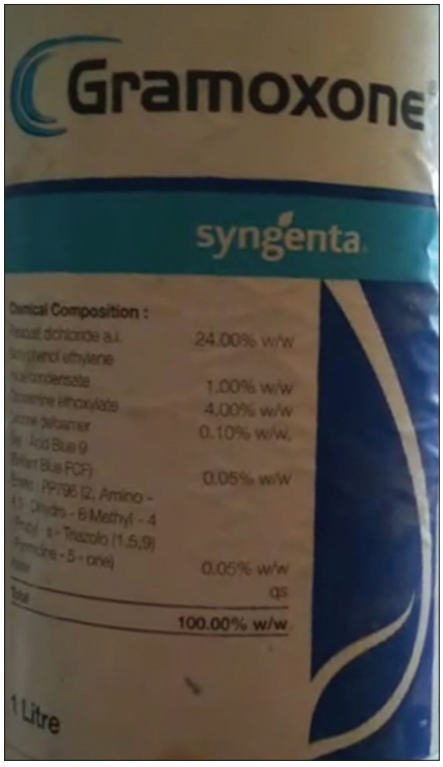

Figure 1: Label on the bottle of the poison ingested by patient 1

Initially, he was kept nil per mouth for 72 and received intravenous fluids. He was allowed clear oral liquids under supervision and gradually received semisolid and solid food. He developed a cough and respiratory distress 7 days following admission, chest X-ray and high-resolution computed tomography of the chest showed fibrosis (Fig. 2).

The boy was treated with oral pirfenidone 200 thrice daily, nebulization with duolin 8 hourly, budecort 12 hourly, and ambroxol syrup. His SpO_2 was 88% on day 7 and improved thereafter. He also received intravenous N-acetylcysteine 1 g over 3 h and 1 g twice daily thereafter, from day 1 of admission, Vitamin E (400 IU) 2 tabs thrice daily. He showed no signs of hepatic and pancreatic injury both clinically and biochemically. He recovered gradually. The psychiatrist's opinion was taken and counseling was done during her stay. He was discharged on day 20 following admission with urea and creatinine values of 15 and 1.2, respectively. Two weeks following discharge, his urea was 28, creatinine 1.1, AST 359, and ALT 218. Signs of liver injury were probably due to paraquat and were treated with ursodeoxycholic acid 300 twice daily for 4 weeks. 6 weeks following discharge his urea was 19, creatinine 0.8, AST 56, and ALT 35. Chest X-ray showed no more progression of fibrosis and the electrocardiogram remained unremarkable.

DISCUSSION

Paraquat acts by generating free oxygen radicals. It has no antidote. The clinical course is dose-dependent. Aggressive decontamination with gastric lavage and adsorbents like activated charcoal (1–2 g/kg) and Fuller's Earth (1–2 g/kg) should be

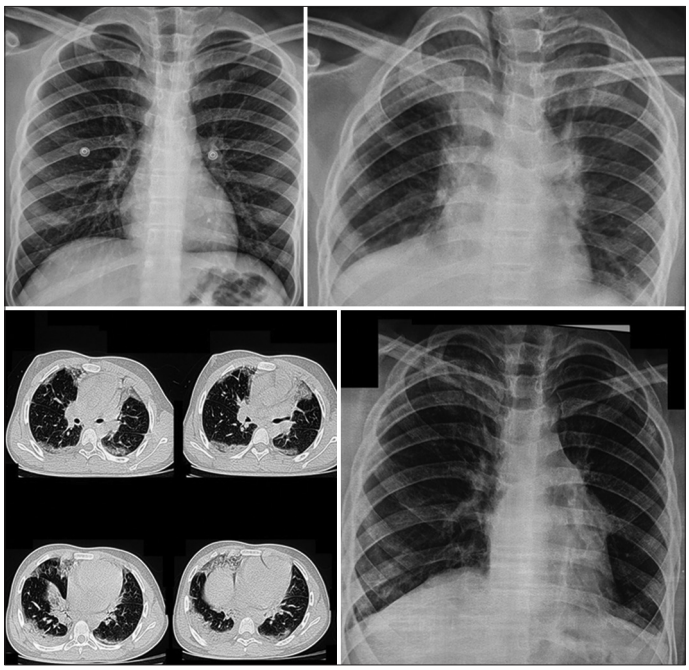

Figure 2: Chest X-rays and high-resolution computed tomography of chest, all of which belong to patient 2. Left upper chest X-ray, taken on admission, is normal. Right upper chest X-ray shows fibrosis, taken on day 7 following admission. Corresponding high-resolution computed tomography of chest taken on the same day. Right lower X-ray showed no progression of fibrosis taken after 6 weeks

instituted as soon as possible (ideally within 1 h) to prevent absorption [4]. Few physicians do not recommend gastric lavage as paraquat is corrosive. As our patients presented late, we did not perform gastric lavage.

Exposure to this poison can be confirmed by a simple and inexpensive urinary dithionite test [4] at the bedside. However, this test was not available to us at that moment. Hemoperfusion within 4 h of ingestion of paraquat has shown to be effective in the reduction of paraquat levels [1]. Hemodialysis has only a supportive role in patients who have developed acute tubular necrosis. Maintaining renal function through hemodialysis reduces plasma paraquat level and in turn minimizes concentration in alveoli. Hence, our patient 1 received hemodialysis and the patient 2's parents withdrew hemodialysis consent after one session but that one session helped to reduce the blood concentration of the toxin.

Anti-oxidants like N-acetylcysteine (150 mg/kg over 3 h; 300 mg/kg over 24 h for up to 3 weeks) have some role by increasing intracellular glutathione (antioxidant) [5]. Both of our patients received N-acetylcysteine. Vitamin C given soon after paraquat ingestion increased oxidative stress [6], so we avoided Vitamin C.

Immunosuppression with glucocorticoids such as methylprednisolone and cyclophosphamide is sometimes used but the benefits of those agents are doubtful [4]. We avoided this strategy after considering the risks and benefits. The outcome depends on both, dose and time to avail medical help. Plasma paraquat concentration indicates prognosis [4]. However, these tests are not available commercially. Young age, percutaneous or inhalational route, exposure to less paraquat, and lesser degrees of leukocytosis, acidosis, renal, hepatic, and pancreatic failures on admission are among good prognostic factors [7]. Both of our patients are young and ingested <50 mL of the poison. Progressive pulmonary fibrosis causes death 2–3 weeks following ingestion. Furthermore, in some cases, patients may have symptom-free intervals of a few weeks, and later succumb to multiorgan failure. Hence, they are under our close and active observation currently.

Loss of renal function contributes modestly to the large increases in creatinine following paraquat poisoning. The rapid rise in serum creatinine most probably represents increased production of creatine and creatinine to meet the energy demand following severe oxidative stress [8]. In both of our cases, there was a rapid rise of creatinine but the normalization of creatinine in the case of patient 2 is explained by this mechanism. Additional biomarkers of kidney injury are required [8].

Frequent monitoring of clinical status and laboratory parameters are the other supportive management, which will help in the early deterioration of the patient's status and hence, early interventions and increased survival [9]. Pesticide action network group showed in a study that farmers buy and use paraquat in an unsafe manner. The literature on paraquat poisoning worldwide as well as in India is very few. The evidence on management is very much non-specific and proof of survival benefit is lacking. Paraquat will continue to have a high mortality rate [10] due to the lack of specific antidotes.

CONCLUSION

With aggressive gastric decontamination, early hemoperfusion and early supportive care may salvage a few patients. There is a need to decrease the use of deadly compounds like paraquat and use alternatives. Paraquat has been banned in 32 countries. In India, only the state of Kerala has banned its use. There is a need for medical personnel to conduct studies on the effects, treatment strategies, and outcomes of this deadly poison. In our cases, both patients were young, ingested small amounts of paraquat, and were treated with hemodialysis early. Hence, they survived. Hence, the outcome of this poison is not all gloomy.

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