



CASE REPORT



<https://dx.doi.org/10.4314/joma.v8i1.6>

PARAQUAT POISONING- CHALLENGES OF MANAGEMENT IN A TERTIARY INSTITUTION IN SOUTH EASTERN REGION: CASE REPORTS

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ABSTRACT

BACKGROUND:

Paraquat (1,1'-dimethyl-4,4'-bipyridylum dichloride) remains widely used as a herbicide in many developing countries, despite being banned in several Western nations. Poisoning from paraquat poses major clinical and public health challenges, especially in resource-limited settings such as sub-Saharan Africa, where access to critical care is limited and mortality rates remain exceedingly high.

CASE PRESENTATION:

This report describes two cases of paraquat poisoning managed in the Children's Emergency Room (CHER) of the Paediatrics Department, University of Calabar Teaching Hospital. Case notes were reviewed and clinical details documented. The patients, aged 12 and 3 years, respectively, presented approximately 24 hours after ingesting "Dragon", a paraquat-based herbicide. Both children exhibited similar symptoms on arrival and had comparable patterns of disease progression.

INTRODUCTION

Paraquat (1,1'-dimethyl-4,4'-bipyridylum dichloride) is one of the most widely used herbicides.¹ It is a quick acting, nonselective herbicide, which is highly toxic for humans and most animals. The extent of poisoning caused by paraquat depends on the amount, route, and duration of exposure and the person's health condition at the time of the exposure. In most

Identified risk factors included storage of the herbicide in familiar household containers, the caregivers occupation (farming), use of inappropriate home remedies before presentation, and delayed arrival at the hospital. Management was significantly constrained by the lack of paediatric ventilatory support and the prohibitive cost of available options. Both children subsequently developed multi-organ failure and died. In the first case, financial limitations further impeded optimal intervention.

CONCLUSION:

These cases illustrate the severe threat posed by paraquat ingestion and the numerous challenges encountered in its management in low-resource environments. There is an urgent need for stricter regulation of hazardous herbicides, improved community awareness, and enhanced critical care capacity to reduce the high mortality associated with paraquat poisoning.

KEYWORDS:

Paraquat, Multi-organ failure, Herbicide, Poisoning, Paediatrics

developed countries, it is used by only licensed operators and measures have been taken to avoid poison being ingested unintentionally.^{1,2}

Paraquat inhibits the reduction of nicotinamide adenine dinucleotide phosphate (NADP) to nicotinamide adenine dinucleotide phosphate hydrogen (NADPH), resulting in the overproduction of reactive oxygen and nitrite species that destroy the lipids of cell membranes.¹⁻³ In the lungs, paraquat is taken up against a concentration gradient which leads to inflammation, leukocyte recruitment, late pulmonary fibrosis, resulting in hypoxemia unresponsive to treatment.^{1,4,5,6}

Following ingestion, pain and swelling of the mouth and throat occurs followed by nausea, vomiting, abdominal pain, and diarrhea which

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could lead to dehydration, electrolyte abnormalities and low blood pressure. Poisoning also occurs as a result of prolonged skin exposure and inhalation.⁷⁻⁹

If a person survives the toxic effects of paraquat poisoning, long-term complications could occur, including lung scarring, kidney failure, heart failure, and esophageal strictures.^{2,10}

Death occurs even when a sip is taken and cause of death is usually from pulmonary involvement.^{1,2}

Paraquat can be mixed easily with food, water, or other beverages when it does not contain the safeguard additives such as dyes, odourants and emetic agents, this poses a challenge in preventing poisoning especially in the children, who are the most at risk. Others at risk include licensed applicators of paraquat.⁵ In developing countries, safety guidelines such as mode of storage, disposal and safeguard additives are not enforced thus poisoning has been associated with drinking of poison put in a water bottle or disposal of empty paraquat containers in areas accessible to the children.⁵⁻⁹

The severity of paraquat poisoning is exacerbated by pre-existing morbidities and may result in mortality. There are so many challenges that bedevil its management especially in resource poor settings like in sub-Saharan Africa particularly Nigeria. Challenges range from the choice of paraquat as herbicide despite its toxic nature, packaging, storage, handling and mode of disposal.⁵⁻⁹ Most developed countries have banned this herbicide or put it under restricted use by professionals. Even in developed countries where it is used, attention has been given to making it more pungent and changing its colour to alert individuals of its toxicity. Unfortunately, it is yet to be banned in most developing countries and guidelines regarding its use are not readily available to farmers.^{2,5-9}

Undocumented cases of accidental exposure causing immense harm to farmers and agricultural workers have been observed. Clinical features of its poisoning following accidental or deliberate ingestion are due to generated reactive oxygen species which cause cellular damage ultimately leading to multiorgan failure with extremely high case fatality.¹¹⁻¹³ Prevention is key in its management but when ingested, treatment is best done immediately using oral administration of activated charcoal or Fuller's earth in order to bind ingested paraquat.

Skin exposure to liquid paraquat will require removal of clothing, washing of the entire body with soap and water.^{11,12}

Hospital management involves nasogastric suction for ingestions that present within 1 hour. Supportive care measures include intravenous fluids, medications to help with breathing and to raise low blood pressure, a ventilator to support breathing, and possibly dialysis for kidney failure. Administration of excessive oxygen should be avoided unless there is worsening cyanosis. No proven antidote or cure exists for paraquat poisoning.¹¹⁻¹⁴

According to a study in Europe,² the incidence of paraquat poisoning was 3.8/100000 inhabitants per year while the median age for the children was 13.4 years.

In a study in Asia,⁴ most of the children were accidentally exposed to paraquat 67.5%, while 32.5% had intentionally ingested paraquat. Most of the children were below three years of age.

In Africa and by extension Nigeria, most studies^{8-9,15-16} were carried out on animals and its impact on agriculture. Few studies gave isolated case reports on the outcome of paraquat poisoning in children. Udensi and Willey⁵ produced a report respectively on the use and impact of paraquat poisoning on the Nigerian population and agriculture with a view to reduction in its use by the populace.

Despite these, in Africa and specifically Nigeria, there is paucity of literature on the prevalence of paraquat poisoning in children. In this article two cases seen at the Paediatric Unit of the University of Calabar Teaching Hospital are presented. This is aimed at bringing to the fore the menace associated with paraquat poisoning and challenges in its management in our environment.

FIRST CASE REPORT

S. D. T. a 12-year-old male admitted into Children Emergency Room (CHER) and spent 3 days on admission.

Patient presented with a history of ingestion of dragon (paraquat) of 12 days duration, recurrent

vomiting of same duration, fast breathing of seven days, fever of five days, abdominal pain of four days, facial and leg swelling of same duration.

Patient was in his usual state of health when he mistakenly drank a chemical stored in a sprite bottle which he mistook for zobo. The chemical was kept in the father's room. Father is a farmer who uses this chemical to clear weeds. patient ingested about 5-10 ml. Few hours following the ingestion of the chemical, patient started vomiting.

Vomiting was said to be non-projectile, approximately 150-200 mL per episode, 1-2 times daily, containing recently ingested meals and one episode of bloody vomitus.

Five days after, the child developed fast breathing which was gradual in onset and progressively worsened. Fever was low grade and continuous while abdominal pain was insidious in onset, mostly felt at the upper part of the abdomen, colicky in nature, severe enough to stop his daily activities, associated with a gradual onset of abdominal swelling. no frequent passage of loose stool.

There was also early morning facial puffiness which regressed as the day went by, with bilateral leg swelling and reduced urine volume. There was no blood in the urine, no reduction in urine volume, no pain on urination, no frequency or urgency.

Following the ingestion of the chemical, the child was given palm oil and coconut water, but with persistence of symptoms, patient was initially taken to a maternity home and 2 other private hospitals prior to his presentation at the General hospital, Calabar, Cross River state, where he was referred to our facility following worsening of symptoms.

On general physical examination, the child was acutely ill-looking, febrile (38.0°C), in respiratory distress, mildly pale with facial puffiness and in severe painful distress, cyanosed, anicteric, not dehydrated, no lymphadenopathy, non-pitting pedal oedema up to the ankle bilaterally, weight on admission was 26 kg.

SpO₂ on presentation was 31% which improved to 55-60% on oxygen.

On systemic examination, the child was dyspneic and tachypneic with respiratory rate of 44 breaths/min. There was tachycardia (124 bpm), blood pressure was 95/59 mm/hg. There was marked tenderness at the epigastrium and right hypochondrium with soft tender hepatomegaly. The child was conscious restless and irritable without signs of meningeal irritation.

A working diagnosis of paraquat poisoning complicated by gastritis and multi-organ failure was made. The child was admitted, and the following investigations were requested: urgent serum electrolytes/urea/creatinine (E/U/Cr), chest radiograph (CXR), abdominal ultrasonography, upper gastrointestinal endoscopy, full blood count (FBC), random blood sugar (RBS), urinalysis, liver function tests (LFT), activated partial thromboplastin time (APTT), international normalized ratio (INR), and serum protein levels.

Intranasal oxygen was commenced due to severely low oxygen saturation. Intravenous fluids (5% dextrose/saline) were administered at maintenance rate. Additionally, Intravenous cimetidine and omeprazole were also given, and urine output was closely monitored.

The Gastroenterology, Respiratory, and Nephrology teams were invited to co-manage the patient, and arrangements were made for admission into the Intensive Care Unit (ICU). The caregivers were counselled extensively regarding the child's condition and guarded prognosis.

Most investigations could not be done due to financial constraints. However, urinalysis performed on the day of presentation was normal. Nutritional support with fortified pap was commenced, along with oral vitamin A (200,000 IU), calcium suspension, intravenous vitamin K, and syrup Astymin.

Despite these interventions, the child's condition progressively deteriorated. He became increasingly irritable, oxygen-dependent, and persistently hypoxic, with oxygen saturation ranging between 42–50% even while on supplemental oxygen. He developed facial puffiness and high-grade fever. Upon removal of oxygen, he became progressively tachycardic, tachypneic, with further deterioration in oxygen saturation.

Seventy-two hours into admission, the child developed repeated gasping respirations and subsequently stopped breathing.

Cardiopulmonary resuscitation was initiated but was unsuccessful. The patient eventually passed on and a request for autopsy was declined by the parents after appropriate counselling.

SECOND CASE REPORT

J.E.E, a two-year-old male, presented with a three-day history of paraquat ingestion and a one-day history of jaundice.

The Child ingested 2mL of dragon herbicide stored in a soda bottle hidden under his father's bed. Following which he began vomiting copiously with vomitus containing recently ingested feeds laced with the colour of the ingested chemical. Two days later he developed jaundice which progressively deepened with associated passage of concentrated urine without a change in stool colour. For the above complaints, his mother gave coconut water, palm oil and enema with warm water following which she took the child to a midwife who gave antibiotics and intravenous fluids. As symptoms worsened the child was brought to our facility where the child was admitted. Examination findings revealed an acutely ill-looking child who was initially not in respiratory distress, not pale or jaundiced, and had normal vital signs. Intravenous fluids and antibiotics were commenced and several investigations were carried out. Twenty-one hours after admission, the child developed respiratory distress, which progressively worsened (SpO₂: 90% in room air). Renal and Liver function test showed markedly deranged values (Aspartate amino transaminase(AST): 128iu/l, Alanine aminotransferase(ALT): 208iu/l, Total Bilirubin: 19µmol/l, Direct Bilirubin 5.1µmol/l; INR was deranged (1.4). Renal function test showed hypokalaemia (2.9mmole/l) and elevated creatinine levels, (180mmole/l) Gastroenterology, CTU and respiratory sub-specialties were called to review. Omeprazole, metronidazole, urso-deoxycholic acid, spironolactone and fat-soluble vitamins were added. The child developed progressively worsening ascites, which revealed straw-colored fluid on abdominal paracentesis. The child continued to deteriorate and was certified dead after five days of admission.

DISCUSSION

Paraquat is highly toxic to humans; one small accidental sip is often fatal and there is no antidote.^{1,2} From the case reports, the children were from two age groups, pre-school and adolescents and both parents were farmers in the rural area.

Most cases of accidental paraquat poisoning occur in the younger age group, while poisoning in older individuals is often associated with suicide. Eddleston et al.¹² reported that most poisonings were self-inflicted, which was not the case in the above reports. However, he noted that such cases occurred more frequently in rural areas, which is consistent with the two cases observed.

Both children in the above case reports accidentally drank the substance from bottles that were not the original containers and were mistaken for edible substances. In our environment herbicides are used by farmers to control weed growth. Unfortunately, strong policies and/or guidelines regarding its use and storage are not made available to them, especially those in the rural areas.^{8-10, 15-16} The parents of both children were farmers, the pesticide was stored in soft drink bottles with colours resembling locally made fruit drinks. Also, these bottles containing these pesticides were kept where it was accessible to the children. In western climes, this herbicide has either been banned, permitted to be used by selected people or strict guidelines are made available as regards its use and storage.^{1,3,4} Also, additives that give a pungent smell are added, and designated bottles are used to prevent accidental ingestion. Unfortunately, this precaution was not observed by the parents in the two cases. In our environment, however, these pesticides are often sold in well-labelled containers but poured into any container provided by the purchasing farmer.⁵

Both children took sips of approximately 2-5 mL. From various literature^{1,2,10,13,15} any quantity of poison ingested into the stomach (from a sip to large quantities) has high mortality rate.

The first aid treatment applied by the parents of both children involved inducing emesis using locally prepared foods, which worsened the symptoms. In most cases, forced emesis is attempted in an effort to remove the offending agent, as seen in the

above case reports. However, this approach has been shown to have minimal benefit and can lead to harmful effects, which can lead to aspiration with necrosis of lung tissue and lung fibrosis.

It has been shown in literature that better prognosis is associated with prompt presentation at the health facility that is trained to manage pesticide poisoning.^{12-14,18,20} Unfortunately, in both cases, there was a delay in presenting at our facility which contributed to the poor prognosis.

In the first case, the history included initial ingestion of paraquat, followed by vomiting, jaundice, and passage of concentrated urine without any change in stool colour. On presentation at our facility 12 days later, vital signs were initially normal, but 21 hours into admission, the child developed respiratory distress, which progressively worsened.

In the second case, the child presented with history of ingestion of paraquat followed by recurrent vomiting then fast breathing, fever, abdominal pain, facial and leg swelling

In most cases, the initial presenting symptoms are first observed in the digestive system². Initial presenting symptoms were essentially the same which included vomiting with the first case having an episode of bloody vomitus. Rahmani et al¹³ corroborated this in their study where the common presenting feature was vomiting, respiratory disorders and kidney dysfunction.

Elimination of paraquat occurs in kidneys within the first 24 hours and continues to the next 100 days in the absence of mortality. Renal involvement occurred in the first case about 3 weeks following ingestion which is in keeping with other reports¹⁸.

The major effect of paraquat follows its accumulation in the lungs with lung cell damage causing decreased gas exchange and respiratory impairment. The lung involvement has two phases: an acute alveolitis over 1–3 days followed by a secondary fibrosis. Most of the patients 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. Studies 2 show that larger volumes ingested (50–100 mL) result in fulminant multiple organ

dysfunction while ingestion of 5–10 mL show predominant involvement of the kidneys and lungs as seen in the two cases. Mortality still remains high irrespective of the amount ingested

In accordance with literature,¹⁷ both patients had respiratory failure in addition to multiple organ damage prior to demise.

Liver involvement is a common feature of paraquat poisoning and was observed in the second case in the course of the illness with progressively worsening ascites which revealed straw coloured fluid on abdominal paracentesis confirming liver involvement for which spironolactone was given. Studies^{2,4,18-20} have shown that high mortality rate is associated with deranged liver function test and EUCr (elevated creatinine and blood urea with hypokalaemia) when compared to the survivors. This could explain why mortality was inevitable especially in the second case who had deranged liver and renal functions. Unfortunately in the first case, derangements in these laboratory values could not be quantified which posed a challenge in the management.

In both cases, various specialties were involved in their management, in keeping with the multi-organ involvement. Unfortunately, late presentation worsened the prognosis in addition to unavailability of paediatric ventilatory support in our facility.

Management is basically symptomatic as adequate antidotes for the poisoning is unavailable in our locale. Omeprazole, metronidazole, urso-deoxycholic acid, spironolactone and fat-soluble vitamins added were used as supportive treatments.

Initial management of paraquat poisoning found to be useful involves lavage of the stomach with activated charcoal. Also, elimination methods such as hemodialysis when done early have been observed to be useful in a few studies. Reduction of toxic levels by use of immunosuppressants like corticosteroid and use of n-acetyl cysteine have been reported. Unfortunately, in the developing world, most patients present late to health facilities, as seen in the above case reports. In addition, the necessary management methods and expertise were unavailable in our setting.

In both reports, there was delay in presentation at the tertiary health facility which worsened

prognosis. Despite the fact that they presented at pharmacies, primary and secondary health facilities, knowledge of appropriate first aid techniques were not applied.

According to the literature, ² oxygen therapy should be avoided as much as possible, as it can worsen lung injury by providing additional substrates for reactive oxygen species generation. Its use is indicated only when oxygen saturation falls below 90%. In the second case, it was administered when the saturation dropped below 60%. Unfortunately, in keeping with other reported cases 16-20 in various regions, both patients did not survive.

CONCLUSION

Globally, paraquat poisoning is associated with high mortality index. These indices are relatively higher in low and middle income countries, like Nigeria. This stems from the lack of expertise in administering first aid in the primary and secondary healthcare settings, delay in presentation to tertiary healthcare facilities and financial constraint. Advocacy should be for proper storage of all poisonous agents and strict regulations by government to control their usage.

ACKNOWLEDGEMENT

We would like to acknowledge all the health care workers that contributed in the management of the above patients.

CONFLICT OF INTEREST:

None

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