Severe Paraquat Poisoning In A Pregnant Teenage Female: A Case Report

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ABSTRACT

Background:
Paraquat (PQ), a highly toxic herbicide, is used extensively in agriculture and poses significant risks of poisoning, particularly through ingestion. Although organophosphate poisoning is more common in Nigeria, paraquat poisoning, often associated with high mortality, remains a serious concern.

Materials and Methods:
This report presents a case of an 18-year-old pregnant female who ingested paraquat in a suicide attempt, leading to fatal respiratory failure. The patient initially presented with mild symptoms but developed severe respiratory distress and multi-organ failure five days post-ingestion.

Results:
Despite early medical intervention and intensive care, her condition deteriorated rapidly, resulting in death.

Conclusion:
The report highlights the severe systemic toxicity of paraquat, the importance of early and sustained medical intervention (as the clinical course is often protracted), and calls for heightened public and healthcare provider awareness regarding its management and prevention.

Keywords: Paraquat, Poisoning, Suicide

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INTRODUCTION

Paraquat (PQ) is a chemical (1,1- dimethyl-4 – 4-bipyridylium dichloride) used in the agricultural sector as an herbicide. Pesticides containing paraquat come in different brands in Nigeria: Dragon, Paraforce, Weed-off, Slasher, Paraquin, and Reliquat.[1] It is highly toxic and a cause of poisoning in humans. Paraquat poisoning can occur incidentally particularly in farmers or intentionally in suicidal attempts. Intentional paraquat poisoning is uncommon, unlike organophosphate poisoning in Nigeria.[2,3] It is associated with high mortality and morbidity.[4,5] Its case fatality is between 33% to 70%.[4-6] The clinical presentation ranges from mild gastrointestinal symptoms to severe multi-organ failure with full recovery or fulminant disease/death outcomes as the case may be.[7] Its management is largely supportive and there is no antidotal therapy.[7] The wide availability and accessibility of this herbicide for agricultural purposes, the high toxicity risk and the lack of an effective antidote call for report of this poison so awareness can be heightened on the health menace paraquat poisoning can cause and its accessibility can be limited.

This report therefore presents a pregnant teenage girl who had paraquat poisoning with suicidal intent and had a fatal outcome from respiratory failure.

CASE REPORT

An 18-year-old female was admitted to the emergency unit of Delta State University Teaching Hospital in Western Delta, Nigeria. This was 6 days after ingesting about 10 mL of a herbicide (Dragon brand: WACOT Nig Ltd, 76G5+QCX, Sabo Lane, Nassarawo 771104, Gombe, Nigeria) containing 24% paraquat, {which she had accessed at home}, an attempt to commit suicide following being jilted by her boyfriend after confirming she was pregnant for him. She presented with a 2-day history of difficulty in breathing associated with a non-productive cough and generalized abdominal pain.

She had developed vomiting and generalized, colicky, burning abdominal pain shortly after ingestion of the herbicide and was taken to the peripheral hospital by her relatives. As of the day of ingestion, 6 days prior to presentation to our facility, she had no shortness of breath or cough. She was managed with activated charcoal, intravenous fluid and antibiotics; and was discharged from the peripheral centre the next day.

Difficulty with breathing started 5 days following poison ingestion, gradually and progressively worsened to occur at rest. No history of chronic cough, asthma, history suggestive of heart failure or any underlying medical disease. She was resuscitated at a nearby hospital for a day and was referred for an intensive care admission.

Physical examination revealed a conscious woman in respiratory distress, with a temperature of 37.5°C, blood pressure of 130/70 mm Hg, heart rate of 110 beats/min, respiratory rate of 32 breaths/minute, oxygen saturation (in room air) was 78% and on oxygen was 88%. Both lungs were clear to auscultation. There was mild generalized tenderness worse on the right hypochondria region, and normal neurological findings. Laboratory investigations showed deranged liver and kidney function tests; LFT -

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Albumin-32g/l, ALP- 176, ALT-31, AST- 28, bilirubin conjugated 15.9, bilirubin total- 22.2 GGT- 27; E/U/CR: Na- 137mmol/L, K- 4.5mmol/L, HCO3- 12mmol/l, CL- 108mmol/L, urea- 224.4mg/dl, Creatinine: 16.82mg/dl; FBC: PCV-25.4%, MCH- 83.8 MCH- 30.4, WBC- 17.11X106/L (Neutrophil count-86.6%, Lymphocyte count- 7.6%), Platelet- 187,000/L; Beta HCG Qualitative- positive; Urinalysis: colour- slightly cloudy& yellow, epithelial cell- scanty (1-5/hpf), protein +, blood +, Haemoglobin +, Leucocyte esterase- trace; HIV Antibody test: Negative; Hepatitis B virus surface Antigen: non-reactive, anti-HCV: non-reactive. An abdominal ultrasound scan showed a 10-week gestational age viable fetus and features of bilateral acute parenchymal disease. A chest X-ray was initially deferred on account of the pregnancy.

She was diagnosed with paraquat poisoning in pregnancy with multi-organ failure (acute kidney injury and acute respiratory distress syndrome). Treatment included intravenous fluid- 5% dextrose saline 8hourly, antibiotics- intravenous ceftriaxone-sulbactam 1.5grams 12hourly, oxygen therapy, work–up for urgent haemodialysis, close monitoring, and consultation with ICU and O&G specialists.

She was admitted to the intensive care unit but despite care here, her condition deteriorated with respiratory rate as high as 60 cycles/min, and patient exhaustion leading to intubation and mechanical ventilation. Post-intubation, her condition fluctuated with periods of instability and unfortunately died from respiratory failure about 12 hours from time of presentation.

DISCUSSION

Poisoning is of public health concern globally as it is associated with high morbidity and mortality.[2,3,4] Poisoning can be from pharmaceuticals, industrial chemicals, pesticides, cosmetic products, and food.[8] World Health Organisation (WHO) accounted 20% of global suicides to be due to pesticide self-poisoning.[9] Organophosphate poisoning has been reported to be the most common pesticide used for self-harm in Nigeria.[2,3,10,11] Organophosphate toxicity, unlike paraquat (PQ) poisoning, is more commonly encountered as a cause of deliberate self-poisoning, especially in Africa and Asia. Its management is well-established, largely due to the availability of an antidote, which has significantly influenced its treatment protocols and outcomes. The public health menace of self-harm poisoning can be likened to substance abuse as both commonly affect young people, and largely result from or result in psychological issues.[12,13]

Paraquat (PQ) is a highly toxic herbicide that can cause poisoning when ingested (as in this present case), in contact with the skin or inhaled. Dermal and inhalation routes have been shown to be far more benign than oral because of less systemic absorption.[4] While accidental poisoning in farmers and contamination of common plants have been well reported and documented in Nigeria,[1,14] that of deliberate paraquat pesticide self-poisoning by direct ingestion is sparse.[15,16] In the United states, a 5-year period review of agricultural and horticultural poisoning revealed that paraquat poisoning accounted for only 0.34% of the over 300,000 cases, but had the highest mortality rate of 13% of all fatal

cases.[17] PQ is however a common agent of poisoning in Asia.[13]

PQ manifests clinically with features of local irritation due to its corrosive property: erosion and ulceration of the tongue, buccal mucosa, oesophagus, and stomach; and systemic manifestations due to its cytotoxic effects by generation of superoxide radicals via redox reaction that damage cells through lipid peroxidation.[7] The primary target organs are the lungs, kidneys and liver manifesting with features of acute respiratory syndrome, acute kidney injury and acute hepatitis respectively. The clinical course is often protracted and depends on the severity which ranges from mild (20 mg/kg ingestion, with gastrointestinal symptoms and full recovery expected), to severe (20-40 mg/kg ingestion, resulting in caustic lesions, renal failure, and fatal respiratory complications within 2-3 weeks), to fulminant (40 mg/kg ingestion, leading to rapid multiple organ failure and death within hours to days).[7] The patient in this case report had a severe form with a fatal outcome. Despite her early presentation to a health facility, she was discharged from the hospital the following day. This could be attributed to poor knowledge of PQ clinical course and thus the need for close clinical surveillance of symptoms that have delayed onset and appropriate counselling of patient and relatives. Adejumo *et al.* in Western Nigeria reported a similar case of a 23-year-old female who developed Acute Kidney Injury (AKI) on the 4th day following PQ ingestion but recovered after 2 sessions of haemodialysis.[16] This case however didn't present with respiratory complication as in the present report. The reports of two similar but fatal cases of paraquat poisoning in India and Nigeria by Raghu *et al*. and Slater *et al*. showed the high risk of mortality associated with cases with respiratory sequelae.[15,18] Bismuth et al. in a systematic review of 28 cases of PQ cases looked at the determinant of PQ poison outcome.[4] They noted that a fatal outcome could occur with even small ingestion of 20% PQ solution, either due to circulatory failure within the initial 3 days or because of gradual and irreversible lung fibrosis within 5 to 31 days. This index case took about 10mls of 24% PQ herbicide (Dragon brand) and manifested features of acute respiratory distress syndrome (ARDS) from the 5th day following ingestion. Other factors reviewed to be associated with severe PQ poisoning by Bismuth *et al.*[4] include ingestion on empty stomachs, presence of gastric and intestinal ulcerations, organic renal failure, and oxygen administration. Similarly, Lee *et al.*[19] studied predictors of survival after acute paraquat poisoning and concluded that young age, exposure through the skin or inhalation, lower levels of paraquat exposure, and milder manifestations of leukocytosis, acidosis, and renal, hepatic, and pancreatic dysfunction upon admission are positive indicators for survival following acute paraquat poisoning.[19]

Treatment of paraquat poisoning is targeted at prevention of drug absorption by decontamination, drug elimination- by gastric lavage, forced diarrhoea, dialysis; and supportive care as there is no specific antidotal therapy.[4,7,20] Supportive therapy involves high-dose antioxidants (vitamin C, E), N-acetyl cysteine, immunosuppressants with high-dose steroids and cyclophosphamide.[19,20] Agarwal et al and JA Liang et al in their studies stated

that methylprednisone and cyclophosphamide may be effective in treating patients with moderate to severe PQ poisoning and not for fulminant cases.[21,22]

While public health awareness is necessary to prevent PQ poisoning, particularly in the aspect of accessibility (storing far from homes, exhausting it when procured for agricultural purposes), education of health care providers particularly non-specialist health care practitioners on its protracted clinical course and management protocol of acute paraquat poisoning may be necessary.

CONCLUSION

Poisoning is a public health problem and thus public health awareness is crucial in preventing PQ poisoning, particularly regarding its accessibility (such as storing it away from homes and ensuring it's fully used when purchased for agricultural purposes). Additionally, educating healthcare providers, especially non-specialists, on the prolonged clinical course and management protocols of acute paraquat poisoning may be essential.

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