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Case Report

Acute kidney injury following paraquat poisoning: An uncommon case of acute toxic nephropathy in Nigeria

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Abstract

1,1′-dimethyl-4,4′-bipyridinium dichloride (paraquat) is a very rare cause of acute toxic nephropathy in Nigeria unlike in Asia-Pacific region. It is one of the most commonly used herbicides which are highly toxic to humans especially when ingested. Paraquat poisoning is associated with high case fatality of up to 52% with pulmonary and renal complications as the major causes of mortality. We reviewed the case notes of a 23-year-old female who was managed for acute kidney injury (AKI) following paraquat poisoning. We present a case of a 23-year-old female who developed nonoliguric AKI that was diagnosed 3 days following paraquat poisoning. She was managed with hemodialysis and had complete renal recovery. Patients with paraquat poisoning have a high risk of developing AKI which may be delayed; hence, renal function should be closely and continuously monitored in them. Early diagnosis and prompt treatment is key to reducing the associated morbidity and mortality.

Key words: Acute kidney injury, 1,1′-dimethyl-4,4′-bipyridinium dichloride, poisoning

Introduction

1,1′-dimethyl-4,4′-bipyridinium dichloride (paraquat) is one of the most commonly used herbicides which is highly toxic to humans especially when ingested. It is a common chemical that is used in deliberate self-harm and suicide attempt in Asia-Pacific region unlike in Nigeria where caustic soda is the major culprit.[1,2] Paraquat poisoning is associated with high case fatality of up to 52%.[3] This may be due to its inherent high toxicity and absence of effective medical treatment.

The severity of clinical manifestations depends on the quantity ingested which may vary from mild (<20 mg of paraquat ion/kg body weight) to fulminant ingestion (>40 mg of paraquat ion/kg body weight).[4] Paraquat poisoning commonly causes local irritative effects on the tongue, oropharnyx, and oesophagus in mild cases and may affect multiple organs especially the heart, liver, kidneys, and lungs in moderate to severe cases. Renal and pulmonary complications are the major cause of mortality in paraquat poisoning.

We report an uncommon case of acute toxic nephropathy in Nigeria. The patient is a 23-year-old female who developed nonoliguric acute kidney injury (AKI) Following paraquat poisoning.

Case Report

A 23-year-old female was referred to the hospital on account of AKI following paraquat poisoning. She was apparently well until 4 days before presentation when

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she drank about 30 ml of herbicide that was paraquat with the intention to commit deliberate harm following a dispute with her husband. She immediately had three episodes of vomiting that was blood stained and estimated Volume was < 500ml. Few hours later, she developed severe pain following an attempt to swallow both liquid and solid meals. There was associated epigastric pain but no change in bowel habit. There was no cough, chest pain nor difficulty with breathing. There was no associated jaundice. She presented in a private hospital that same day where she was placed on intravenous fluids, antacids and was subsequently discharged home the next day. She, however, presented in a state secondary health facility 2 days after initial discharge on account of worsening odynophagia.

There was no history or physical examination finding suggestive of systemic features of paraquat toxicity following evaluation. However, her renal function test was noticed to be deranged despite being not oliguric. She was treated with antibiotics, antacids, intravenous fluids, and nasogastric tube was passed for feeding. Her renal function, however, worsened after 24 h of admission necessitating her referral.

At presentation, she was fully conscious, oriented, not in distress, well hydrated, not pale, anicteric, and without pedal swelling. Her pulse was 96 beat per minutes, regular, normal volume, blood pressure was 130/60 mmHg, and heart sounds were normal. Respiratory and abdominal examinations were essentially normal. Urinalysis showed pH of 6.0, the specific gravity of 1.020, protein, glucose, and blood were negative while microscopy showed granular cast.

Investigation Results

White cell count was 3600 cells/mm^3 with the normal differential count, hematocrit was 37%, and platelet count was 140,000 cells/mm^3. The liver function test, renal, and liver scan were essentially normal.

She was given parenteral antibiotics, steroids, and proton pump inhibitors. She had two sessions of hemodialysis with complete renal recovery [Table 1]. She also had persistent hypokalemia that resolved with potassium replacement. She was also evaluated by a clinical psychologist and co-managed with a cardiothoracic unit on account of corrosive esophagitis with improvement in Her clinical condition.

Table 1: The renal function profile of the patient

Number of days after paraquat ingestion	Day 3	Day 4	Day 5	Day 6	Day 7	Day 13	Day 22
Urea (mmol/L)	11.4	19.9	32.3	11.5	3.9	3.7	1.9
Creatinine (μmol/L)	450	584	747.1	549	126.1	89.4	76.4
Sodium (mmol/L)	131	137	140.6	136.9	136.8	128.8	136.8
Potassium (mmol/L)	3.3	2.8	3.5	2.6	2.5	2.0	4.3
Bicarbonate (mmol/L)	21	22	20.9	24.7	22.7	27.8	22.9
Chloride (mmol/L)	99	105	112.3	104.8	105.1	91.6	10.7

Discussion

Paraquat poisoning as a cause of acute toxic nephropathy is rare in Nigeria. In previous reports from different parts of Nigeria on causes of acute toxic nephropathy, paraquat was not a commonly indicted etiological factor.[5,6] Delibrate self-harm from toxic chemicals have been reported to be more common in young individuals with difficulty in interpersonal relationships as the major cause.[2]

Paraquat is highly toxic as seen in this patient who developed severe AKI following ingestion of about 30 ml. It is rapidly absorbed after ingestion and distributed into the organs especially the liver, kidney and lungs where it commonly produces its toxicity. Its excretion is primarily by the kidneys in a biphasic manner; early rapid phase and a late, slow elimination phase which may be possibly due to renal tubular damage caused by paraquat itself. This may also partly explain the delayed onset of AKI in some patients with paraquat poisoning as seen in this patient.

The kidneys are commonly affected in paraquat poisoning. Kim *et al*. reported that the prevalence of AKI and acute kidney failure following acute paraquat intoxication was 51.4% and 34.7%, respectively.[7] The quantity of paraquat ingested was also reported to be the most significant predictor of developing AKI following poisoning.[7] AKI may result from direct toxicity on the kidneys. This mechanism of renal injury involves generation of free radicals causing lipid peroxidation, mitochondrial toxicity, activation of nuclear factor kappa culminating into apoptotic death of the renal tubular cells.[8] It commonly affects the proximal part of the tubules where it may sometimes manifest as Fanconi's syndrome.[8,9] Assessment of proximal tubular function which is required to confirm this was not assessed in the patient. However, the indirect mechanisms of renal injury observed were volume depletion, sepsis and multiple organ failure that may have occurred as a complication of the poisoning. Hypokalemia may also occur due to renal tubular acidosis or increase urinary loss of potassium. The index patient had hypokalemia that resolved with potassium replacement.

This patient presented with nonoliguric AKI, which was consistent with previous report,[9] although some patients may exhibit the oliguric type.[10] Diagnosis of AKI could easily be missed if physicians depend solely on urine output to assess renal function in high-risk patients such as in this patient. This therefore reiterates the need to monitor renal function biochemically in such

patients. Dialytic therapy in this group of patients is mainly supportive because dialysis does not remove paraquat from the systemic circulation.

The overall outcome of this patient was favorable possibly because of the prompt diagnosis and treatment of the AKI as well as absence of poor prognostic factors such as leukocytosis, multi-organ failure, older age, ingestion of more than 30 ml, severe acidosis, and early need for intensive care.[11,12]

Conclusion

Patients with paraquat poisoning have a high risk of developing AKI which may be delayed; hence, renal function should be closely and continuously monitored in them. Early diagnosis and prompt treatment are key to reducing the associated morbidity and mortality. There is also need to restrict the accessibility of this potentially harmful chemical to only licensed applicators by the government.

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Conflicts of Interest

There are no conflicts of interest.

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