Response:
Accidental Potassium Bromate Poisoning Causing Acute Renal Failure
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Response:
I have read with keen interest the case of accidental ingestion of potassium bromate by a two year old boy reported by Adeleke and Asani.1 I sincerely congratulate them for their high index of suspicion of potassium bromate poisoning and for successfully managing the patient amidst inadequate facilities in Nigeria to manage such a case. However, I would like to raise a few points about the history and management of the patient.

Even though the body temperature was not reported, vomiting, diarrhoea, abdominal pains, lethargy and hepatosplenomegaly, as initially presented by the patient, are symptoms and signs of malaria or inflammatory gastroenteritis. Perhaps this was the reason for the initial diagnosis of acute diarrhoea with some dehydration made by the authors. The degree of dehydration of the patient at diagnosis of acute diarrhoea was not reported, however, the passage of frequent and loose wetty stools with weakness may indicate moderate to severe dehydration. Moderate to severe dehydration may result in acute renal failure.4

There is a high possibility of the child ingesting potassium bromate especially when the mother rinsed his mouth with water. However, the quantity swallowed may be small and this may perhaps explain the mild symptoms and signs initially presented by the child. The quantity of potassium bromate ingested by children from previous reports was very small5,6 compared to adults7,8 and this have accounted for the relatively mild symptoms observed in the children. Hepatosplenomegaly has not been reported in previous cases of acute potassium bromate poisoning but this may be caused by a massive intravascular haemolysis. Massive haemolysis and thrombocytopenia may be seen in children with bromate poisoning1 but were not present in the patient reported by Adeleke and Asani.1 However, mild haemolysis was reported as evident by anaemia (PCV- 28%). Thus, malaria or inflammatory gastroenteritis may explain the hepatosplenomegaly reported in the patient. It might have been useful to do complete blood count, blood film morphology and malaria parasites study of the patient. Electrolyte and urea are very important in patients with moderate to severe dehydration which should have served as early pointers to acute renal failure but unfortunately they were delayed until the 4th day of admission.

The mechanism of bromate toxicity is not clearly understood but it has been proposed that renal failure could result from direct tubular toxicity due to induction of active oxygen radicals9, reduced renal perfusion from dehydration and possibly decreased vasomotor tone.10,11 Haemolytic anaemia with haemoglobinuria may also play a role.12 Asymptomatic phase of a few hours may follow bromate poisoning before overt renal failure develops and anuria is usually apparent within 1-2 days of bromate ingestion1 similar to the onset of anuria in the patient reported by Adeleke and Asani.1 Perhaps, a urethral catheterisation would have been more appropriate to monitor urinary output rather than suprapubic needle aspiration performed by the authors.

The authors have centred their discussion on the management of acute bromate poisoning with reference to animal studies which could not be extrapolated to human and may not be very relevant in the clinical management of the patient. The principles of management of acute bromate poisoning are largely empirical and supportive. They include reducing bromate to less toxic bromide ion with specific antidotes. 10% sodium thiosulphate solution, 10- 50 ml (0.2- 1 ml/kg) i.v has been successfully used as a specific antidote in the treatment of acute bromate poisoning in children.5,6 Methaemoglobinemia may require the use of methylene blue.8 In the early stage, patient can be decontaminated with sodium bicarbonate (baking powder) to prevent formation of hydrobromic acid in the stomach9; it might have been worth a trial in this patient. Large recent bromate ingestion may require gastric lavage with a 2% sodium bicarbonate or activated charcoal.10,11 Peritoneal dialysis was performed in the patient not only to reverse the renal failure but to enhance bromate ion elimination since bromates are primarily excreted renally.8

Tinnitus and irreversible sensorineural deafness may complicate bromate poisoning and may be delayed for several days in children or may go unnoticed.9,10,11 Thus, audiometry at presentation and follow-up was very important in the management of this patient. Polynuropathy, characterised by severe burning pain to the leg, is an unusual symptom seen in bromate poisoning in adult and may present very late;
although it may resolve spontaneously after a month. Thus, a neurological examination should have formed part of the follow-up evaluation of this patient.

References:


