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LETTER TO THE EDITOR

Renal Failure and Haemolysis in a Two-Year-Old Child due to Black Water Fever Or Naphthalene Poisoning

Insuffisance Rénale et Hémolyse chez un Enfant de 2 ans: Fièvre de l'eau Noire ou Empoisonnement au Naphtalène?

Malaria remains a major public health problem in the world, with sub-Saharan Africa accounting for about 80% of malaria cases.[1] Black water fever (BWF) is one of the severe forms of malaria. [2, 3] It is a clinical syndrome characterized by severe intravascular haemolysis, haemoglobinuria and acute renal failure, commonly seen after receiving quinine. [2,] There have been reports of haemolysis occurring in patients treated with artemether-lumefantrine combination therapy.[3] There is also a strong association of haemolytic anaemia with naphthalene poisoning, first described in 1949,[4] . In this initial report four Negro infants with mothballs poisoning, presented among others with severe intravascular haemolysis, haemoglobinuria and acute renal failure.

Our patient, BJK, a 2-year- old male child presented with complaints of fever, diarrhoea, vomiting and passage of dark coloured urine associated with reduced urine output for three days. Child was treated with artemether–lumefantrine and paracetamol. His mother gave a history of accidental mothball ingestion, for which vomiting was induced by administering palm oil, peak milk and abdominal compression; the child vomited one ball of the naphthalene. Examination revealed an acutely ill looking child who was conscious with a temperature of 38.5°C. He was pale and jaundiced but other vital signs were stable. His urine coca-cola coloured.

A diagnosis of severe malaria presenting as severe anaemia and black water fever was made. The differential diagnosis was haemolytic anaemia secondary to naphthalene poisoning. The patient's packed cell volume (PCV) was 18% and malaria parasite was positive. Urine microscopy showed no red blood cells or red cell cast. Urine culture showed no bacterial growth. The results of a full blood count were as follows: haemoglobin, 5.8 g/dl; WBC, 9.0×10^9/L; platelets, 220×10^9/L; reticulocytes, 1.5%; neutrophils, 61%; lymphocytes, 30%; monocytes, 2%; and eosinophils 7%. His G6PD activity was within normal limits. The liver function tests were normal except for high bilirubin. The patient was also negative for hepatitis B and C screening. Serial serum electrolytes and urinalysis results are shown in the Table. The index patient improved tremendously after he was transfused with blood and was placed on artesunate.

Table 1: Serial Results of Serum Biochemistry and Urinalysis

	Day 1	Day 3	Day 5
Serum Electrolytes			
Sodium mmol/l	145	150	140
Potassium mmol/l	5.8	5.l	4.2
Chloride mmol/l	110	100	100
Bicarbonate mmol/l	15	20	25
Urea mmol/l	20	8.8	3.8
Creatinine µmol/l	135	87	44
Urinalysis			
Specific Gravity	1.030	1.020	1.020
pH	6.5	7.0	8.0
Glucose	Negative	Negative	Negative
Protein	+++	++	+
Ketone	+++	Negative	Negative
Haemoglobinuria	++	+	Negative
Nitrite	Negative	Negative	Negative
Leucocyte esterase	Negative	Negative	Negative
Bilirubin	+	+	Negative

Protein +, 30 mg/dl; bilirubin +, 15 mg/dl; haemoglobinuria +, 10 mg/dl.

The clinical presentation of BWF is typically with intravascular haemolysis[2] associated with fever, headache, vomiting, malaria parasitaemia and later passage of coca-cola coloured urine, jaundice and renal failure. Our patient had similar presentation except that he never had quinine, halofantrine or mefloquine. The most likely trigger in our patient might be the artemether-lumefantrine he was given on outpatient basis. This corroborates with a report by Aloni *et al*[3] where haemolytic crisis of BFW followed artemether-lumefatrine intake.

The index patient improved tremendously after he was transfused with blood and was placed on artesunate. Oguche *et al* [2] had similarly treated their patients with BWF using artesunate. Our patient's early signs of renal failure were reversed by renal challenges. It is conceivable that other drugs and chemicals can trigger intravascular haemolysis with subsequent development of haemoglobinuria, jaundice and renal failure especially if they are G6PD deficient.[4, 5] This may be the case in our patient, since he had ingested naphthalene ball, but G_6PD activity was within normal limit. However, the laboratory report suggesting normal G6PD activity may not be unusual in the black African type G_6PD deficiency where, false negative G_6PD assay may occur.

The challenge here is that we had no reagents to do the susceptibility test of the blood of index patient to naphthalene to see wither the haemolysis could be attributed to naphthalene or not. Massive haemolysis and haemoglobinuria had been reported in a patient with SCA, [5] however the index patient's genotype was AA. Haemoglobin precipitation in the kidney can lead to renal impairment. This might have been the case in the index patient.

It is very important for physicians who manage cases of malaria to be aware that severe intravascular haemolysis can follow artemether-lumefanthrine administration; and that naphthalene poisoning may mimic BWF and G6PD deficiency.

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