Blackwater Fever in an 82-Year Old Nigerian Woman: A Case Report
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Abstract
Blackwater fever is acute intravascular haemolysis which occurs in some persons with Plasmodium Falciparum Malaria that were treated with Quinine Sulphate. It frequently occurs in children and is usually seen in non-immune or semi-immune individuals. The case being presented occurred in an 82-year old Nigeria woman who did not have acute malaria attack neither did she admit to taking quinine orally or parenterally.

Introduction
Though malaria cases constitute the bulk of cases handled in Private Medical Practice outpatient department, it is only occasionally that you encounter black water fever cases and then it occurs usually in children in African population. Black water fever derives its name from darkish red to brown urine passed by patients who are under attack of Plasmodium Falciparum infection. It is a dreaded complication of malaria fever in non-immune or semi-immune individuals who were treated with quinine preparations. It typically presents with passage of darkish brown or coca-cola coloured urine in addition to the usual features of malaria such as fever, headache, nausea and vomiting as well as abdominal pain. It also occurs in children suffering from Glucose-6-Phosphate Dehydrogenase deficiency.
Black water fever has been described as a complication of treating severe malaria with quinine; antimalarials which are derivatives of amino-alcohol family such as halofantrine and mefloquine have also been implicated in causing blackwater fever.

Case Report
EA is an 82 year old woman who was brought to our hospital with a history of sudden collapse at home and passing darkish brown urine on 31/5/10. It took about 5 minutes before patient was revived and there were no associated convulsive attacks. Patient admitted to having taken herbal medications the previous day for some vague complaints of tiredness and malaise. There was no previous history of passing darkish brown urine. She did not admit to having taken any quinine, mefloquine or halofantrine preparations. Sample of urine voided and shown to doctor was reddish brown in colour. On examination, she was conscious, pale, afebrile with tenderness in her left flank. Her blood pressure was 180/90 mmHg, temperature was 36.20 C. Her liver and spleen were not enlarged. Laboratory tests revealed a PCV of 18%, Malaria parasite blood smear [Thick smear] was negative, WBC count was 5.0x10^9 /l, Neutrophil 46%, Lymphocytes 45%, Monocytes 4%, Eosinophils 5%. Patient was admitted, given artemether 160mg I.M stat, and 80mg LM daily thereafter for 4 days and received a pint of blood. Hydrocortisone 100mg I.V q 6hrly for twenty- hours was given and prednisolone 5mg t.i.d for two days. She was asked to discontinue the herbal preparation she was on previously. Patient’s response was prompt as the urine colour lightened gradually over a matter of days. PCV done 24 hours later was 26% while at discharge 3 days later it was 24%. Renal function tests showed Blood urea of 7.0mmol [Normal range of 1.7-9.1] and a Creatinine of 105 umol/l [Normal range of 50-110umol/l]. Repeat malaria parasite smear test was negative. Patient recovered quickly and was sent home with haematinics.

Discussion
Blackwater fever arises from massive intravascular hemolysys which leads to haemoglobinemia and haemoglobinuria. It has been postulated that malaria parasitized red blood cells undergo antigenic alteration that make them susceptible to immune mediated destruction in the spleen due to the production of auto antibodies against such cells. This derangement could affect non-parasitized red cells thus leading to severe anaemia^2. When
haemoglobin precipitates in the kidney, renal impairment can result and this would lead to rise in blood urea and creatinine levels.

Quinine has been implicated in the pathogenesis of blackwater fever; the incidence of blackwater fever declined from 1950 to 1990s corresponding to the time of emergence of Chloroquine as a drug of choice for treating malaria. Following increasing resistance to chloroquine in malaria therapy and the resurgence of use of quinine in treating resistant malaria, more cases of blackwater fever are being encountered 3,5,7.

Immunity wanes with age and this could be responsible for the patient not having other features of the condition other than passing darkish coloured urine. Her advanced age reduced her tolerance for hypoxia caused by acute hemolysis and this made her collapse. The patient after transfusion never experienced fainting attacks thereafter. Her prompt response to artemether suggests that malaria parasites were the immediate cause of the hemolysis.

Blackwater fever is experiencing a resurgence in regions where malaria is endemic and this has been attributed to use of quinine in treating malaria. A large number of cases of blackwater fever were recently reported in Senegalese4 and Burundi children6.

There is need to look out for this complication in patients who are on quinine therapy for resistant malaria cases.

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References


