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Case Report Open Access

Spontaneous Expulsion through the Anus of *Ascaris Lumbricoides* in a Child with Cerebral Malaria

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Abstract

Ascariasis with malaria is both endemic and prevalent in tropical settings settings and their co-infections should be expected. Asymptomatic ascariasis infection is often missed and untreated. We report a fatal case of cerebral malaria with ascaris co-infection in a 2 year old boy. The diagnosis of intestinal helminths would have been missed, but for the extrusion of several round adult worms out of the body through the anus. The implication of the extrusion of the helminths from the body through the anus is highlighted. Suggestions that may help improve case management of ascariasis in paediatric admissions are also proffered.

Keywords: Paediatric; Intestinal; Parasitoses; Admissions

Introduction

Intestinal parasitic infections and malaria are sources of international concern. Globally, approximately two billion individuals are infected with helminths, while 300-500 million people are infected with malaria annually [1-3]. Most of the infections with malaria or infestations with helminths occur in the sub-Saharan Africa and this setting is endemic for both infections. Nigeria is the most populous country in the Sub-Sahara of Africa. It has a population of 149 million people and a surface area of 356,699 miles with a resultant density of 370 persons/mile [4]. Thus Nigeria is the most populous nation in Sub-Sahara Africa with the highest populace exposed to endemic diseases such as malaria and helminthiases.

A prevalence estimate of 55 million has been reported for ascariasis in Nigeria. More than half of the infections occurs in children [2]. The tropical weather coupled with high levels of poverty, poor hygiene and ignorance of health promotion all favour the transmission of soil transmitted helminths.

More than 50% of the Nigerian population is estimated to have malaria annually. One hundred and thirty two billion naira has been stated to be incurred annually either directly, for the control and treatment of malaria or indirectly as the amount of money lost from inability to work as a result of malaria infection [2]. Children are more susceptible to malaria infections because of the poorly developed immune response to malaria.

Similarities in the geographical predilection by both intestinal parasites and malaria in Nigeria is likely to favour the co-occurrence of both infections in these settings. Reports from Nigeria on the association of malaria and intestinal helminths are however scarce. There is therefore a need to study the association between both diseases. The improved understanding of the association between the diseases might lead to a better management and control of both malaria and intestinal helminthiases.

Case Report

A two year old boy was admitted to the children emergency unit of the Ladoke Akintola University of Technology Teaching Hospital, Osogbo on account of intermittent fever noticed about a week prior to presentation. Convulsions were observed five days prior to presentation. The patient had convulsed five times before presentation and the first noticed convulsion was generalized and lasted 15 mins. The patient had not regained consciousness till presentation. He has also had similar convulsions lasting for less than a minute on four consecutive days. The patient had not accepted any feed since the day he lost consciousness. The amount of urine produced had markedly reduced on the day the child presented. Vomiting of recently ingested feeds which later became bloody was observed a day prior to presentation.

Both parents of the child are 27 years old and tailors. They live in a village and have access to potable water. They have two other children that are well.

On examination the child was unconscious, mildly pale and afebrile with a temperature of 36.3°C. He was well hydrated and had a capillary refill time less than 2 seconds. The patient weighed 12 kg. There was no cyanosis or icterus.

The central nervous system examination revealed an unconscious child with a Glasgow coma score of 6. The occipito-frontal conference was 49.1 cm. Kernigs and brudzinski signs were negative. Hypertonia and hyperreflexia were elicited in the upper limbs, while the tone and reflexes were normal in the lower limbs.

The cardiovascular system examination revealed normal volume and regular pulses at a rate of 116 beats per minute. The blood pressure was 94/60 mm/Hg and only the first and second heart sounds were heard. Examination of the respiratory system and the abdomen were essentially normal.

A tentative assessment of cerebral malaria was made and the full blood count examination on admission revealed a packed cell volume of 29 percent, total white blood count of 6,500 mm^3 with neutrophils, lymphocyte and eosinophil differential counts of 73, 26 and 1 percent

respectively. A platelet count of 371,000 mm^3 was recorded. The cerebrospinal fluid microscopy and chemistry were essentially normal. Trophozoites of *Plasmodium falciparum* were detected in the blood film however. The random blood sugar and the pulse oximetry at admission were normal. The possibility of septicaemia complicating severe anemia was entertained because of the dominance of neutrophils in the white cells.

The tentative diagnosis of cerebral malaria was still left in view. The patient was therefore started on 1 g of intravenous ceftriaxzone daily and intravenous artesuntae to manage the cerebral malaria. The convulsions were treated with daily administration of 60 mg of phenorbabitone and intramuscular paraldehyde when necessary. Although a nasogastric tube was passed for feeding, the child was not fed because the tube kept draining blood and so a diagnosis of upper gastrointestinal tract bleeding was made. Intravenous fluid was administered at maintenance rate for calorie and adequate hydration.

The condition of the child became worse after 24 hours of admission with the development of facial puffiness and deepening coma evidenced by a Glasgow coma score of 3. Fluid input and output were 1010 and 160 mls respectively. Thus the output was 0.56 ml/kg/hr. Round worms were noticed to be migrating out of the anus. A picture of the round worms exiting the anus is found in Figure 1. A diagnosis of Ascariasis and acute kidney injury was therefore made. The electrolytes, urea and creatinine were requested. The prothombrin time, partial thromboplastin, urinalysis and random blood sugar were also requested. The results of the urinalysis was essentially normal, while the results of the other tests are displayed in Table 1.

Figure 1: Round worms exiting the anus

The bleeding from the upper gastrointestinal tract persisted for 48 hours and there was no real improvement in the sensorium, mucosa and pallor was still mild in the patient. There was no marked difference in the amount of urine produced. Therefore the electrolytes, urea and creatinine were re-requested. The results of the electrolytes, urea and creatinine are also displayed in Table 1.

On completion of the course of intravenous artesuate, the blood film examination by microscopy still revealed the presence *Plasmodium falciparum* in the peripheral blood and the patient was commenced on intravenous quinine at 10 mg/kg body weight 8 hourly. At 72 hours of admission the patient went into cardio-respiratory arrest, which was managed by ambu-bagging and cardiac massage. While on the first course of quinine infusion the patient developed hypoglycaemia with a random blood sugar of 1.6 mmol/l which rose to 2.1 mmol/l while on the second course of quinine infusion. Both episodes of hypoglycaemia were corrected with infusion of 4 mls/kg of 10% dextrose water. The strength of the quinine containing dextrose was also increased from 10% to 12%. Three other cardiopulmonary arrests were recorded and they were managed with cardiac massage and ambu-bagging. Consequently the child was transferred to the intensive care unit and placed on a ventilator and intensive care. However clinical condition of the child did not significantly improve and the patient died after 12 hours of care at the intensive care unit. The parents refused to consent to our autopsy request.

At 48 hours		
	Results (mmol/l)	**Laboratory reference value (mmol/l)**
Sodium	126	120 – 140
Potassium	3.5	3 – 5
Bicarbonate	23	20 – 30
Chloride	92	90 – 110
Urea	22.8	1.7 – 9.1
Creatinine	Not done	60 – 120 umol/l
At 72 hours		
Electrolytes and urea	**Results (mmol/l)**	**Laboratory reference value (mmol/l)**
Sodium	132	120 – 140
Potassium	4.2	3 – 5
Bicarbonate	18	20 – 30
Chloride	102	90 – 110
Urea	30.9	1.7 – 9.1
Creatinine	450	60 – 120 umol/l
Clotting test		
PT	37//	14//
PTTK	53//	33//
INR	2.7	

Table 1: Serum electrolytes, urea and creatinine

Discussion

The co-occurrence of malaria and soil transmitted helminths is believed to be common in settings endemic for both diseases. Previous studies also show that these co-infections are common in Africa [5-6]. The child in the present report presented with cerebral malaria which is a severe manifestation of malaria. Previous reports however show that there is no association between severe malaria and ascariasis. A strong association between uncomplicated malaria and intestinal helminthiases has however been previously documented [5-6].

Ascaris lumbricoides infestation is common in pre-school children located in rural areas probably due to the poor sanitary condition and low socio-economic status of most rural dwellers [7]. The helminth has been located in the lungs, intestines and pleural space, but spontaneous migration out of the body through the anus is yet to be reported though the anus is the natural passage route for expulsion in the cycle of the worm and post administration of anti-helminthics. Migration of the adult worms from the jejunum, to the trachea and lungs has been reported to be part of the normal life cycle [8]. Exit to the exterior through a perforated tympanitic membrane and through the pulmonary tissue into a chest tube has been reported [8,9]. Anecdotal beliefs in clinical practice suggest that adult worms migrate from their normal intestinal habitat whenever conditions are unconducive for thriving. A very high fever is believed to be the most common factor making intestinal parasitism unconducive by helminths. This child did not have a high fever and the highest temperature recorded in the present case was 37.5°C.

Could the hypoglycaemia, accidocis and uremia recorded in this child conceivably have constituted unconducive conditions for *Ascaris lumbricoides* in the intestines? Or could there have been other physical or biochemical changes in the body unconducive to worms such that when this child's condition became critical about 24 hours before death the worms took their exit? On the other hand can the exit of the intestinal worms from the body be taken as signs prognosticating impending death just as some reflex activities like micturition and effortless vomiting may sometimes be herald death in critically ill patients.

The diagnosis of ascariasis was accidental in this patient and it is quite possible that the intestinal worms might have contributed in a more significant way to the death of this patient. For instance we do not know the magnitude of the worm infestation and the location where the other intestinal worms might have migrated to. These factors may contribute significantly to morbidity or mortality. An autopsy might have shed more light on these gray areas, unfortunately it was not done.

It is concluded that asymptomatic cases of intestinal helminths parasitoses may be missed. Routine examination of stools of admitted children for ova and intestinal parasites should therefore be the rule in all endemic areas for helminths in non-symptomatic cases at discharge. A combined controlled intervention directed at intestinal helminths and malaria is suggested again as a promising intervention for controlling both diseases [10]. Better planned studies need to be conducted on the association between intestinal helminths and malaria.

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