

Cerebral Malaria Complicated by Blindness, Deafness and Extrapyraxidal Tract Manifestation

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Abstract

Cerebral malaria is a severe manifestation of a parasitic infection caused by *Plasmodium falciparum*. The sequelae of this disease such as blindness, deafness, loss of motor function could be emotionally traumatic and physically disabling. We, therefore, present this case of an 8-year-old boy who presented with high-grade intermittent fever associated with multiple convulsions and prolonged coma. He regained consciousness after 12 days of treatment with intravenous quinine but was found to have blindness, sensory-neural deafness and extrapyramidal sign. This extrapyramidal sign regressed following treatment with chlorpromazine. He also regained his sight and auditory function before he was discharged though not completely. This report is aimed at emphasizing these rare complications of cerebral malaria as well as reminding clinicians working in malaria endemic areas of the world on the need for early diagnosis and prompt treatment.

Keywords: Blindness, Cerebral malaria, Coma

Introduction

Malaria is a parasitic disease affecting about 1 billion people globally and causing about 1.24 million deaths annually especially in the developing countries.^[1,2] About 1% of the patients with *Plasmodium falciparum* develop severe manifestations culminating in multi-organ failure.^[3] Cerebral malaria is one of these severe manifestations with its attendant sequelae such as cerebral palsy, cortical blindness, sensory-neural deafness and rarely extrapyramidal manifestations.^[3-6] Diagnosis of cerebral malaria requires demonstration of asexual form of *P. falciparum* in peripheral blood smear, in thick and thin blood smear films stained by Giemsa stain.^[4] The histopathological hallmark of cerebral malaria is engorgement of cerebral capillaries and venules with parasitized red blood cells (PRBCs) and non-PRBCs.^[5] This case report is intended to remind clinicians working in malaria endemic areas of the world that the disease is still deadly.

Clinical Presentations

An 8-year-old child presented in the children emergency room of the Federal Teaching Hospital, Abakaliki, Nigeria, with a 5 days history of high grade intermittent fever and a 3 days history of multiple, generalized convulsions associated with loss of consciousness. Examination of the patient on admission revealed a comatose child with an axillary temperature of 39°C and pallor. The child had hypertonia and hyperreflexia in all the limbs, and the pupils were dilated and reacting sluggishly to light. The liver was enlarged 3 cm below the right costal margin. Lumbar puncture done after a dose of mannitol yielded clear and colorless cerebrospinal fluid that was not under pressure, and its analysis and culture were normal. Blood film for malaria parasite was positive for asexual form of *P. falciparum* with a density of 29,920 parasites/mm³. Cranial computerized tomography was not done because of unavailability of the facility. A diagnosis of cerebral malaria was made, and the patient was commenced on intravenous quinine and parenteral paracetamol. He was transfused while on admission in the emergency room. The patient regained consciousness after 12 days of treatment but was found to have blindness and deafness following reviews by ophthalmology and ear, nose and throat teams respectively. Extrapyraxidal tract manifestations evidenced by purposeless involuntary movements of the limbs and biting of the fingers

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DOI:
10.4103/2141-9248.160183

were documented by the pediatric neurology team. Based on the above neurological features, the patient received prednisolone for the blindness. He was subsequently placed on chlorpromazine when diazepam could not stop the involuntary movements, and the symptoms subsided markedly after 3 days. He was discharged from the hospital after 30 days of admission following stable clinical condition though blindness and deafness have not resolved completely.

Discussion

Several hypotheses have been documented to explain the pathophysiology of cerebral malaria. These hypotheses include cytoadherence of parasitized red cells,^[7] up-regulation of vascular endothelial ligands.^[8-10] These hypotheses are said to be responsible for clinical features of malaria such as coma, convulsions, and neurological deficits. Sensory-neural deafness in this patient may be argued to result from quinine effect. However, it is not common to recover from deafness due to quinine. This patient recovered significantly before discharge implying that the deafness could be a complication of cerebral malaria that the child suffered. This was also confirmed by the ophthalmologist when fundus examination was done.

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How to cite this article: Manyike PC, Okike C, Onyire NB, Chinawa JM, Austin -Abu JU. Cerebral malaria complicated by blindness, deafness and extrapyramidal tract manifestation. *Ann Med Health Sci Res* 2015;5:321-2.

Source of Support: Nil. **Conflict of Interest:** None declared.