

## Case Reports

# Expressive aphasia: An isolated and reversible complication of cerebral malaria in a child

B. Maini, R. Narayan, A.K. Bhardwaj & P.D. Sharma

Department of Pediatrics, Maharishi Markandeshwar Institute of Medical Sciences and Research, Mullana, Ambala, India

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Cerebral malaria is associated with neurological sequelae in some survivors, especially in children<sup>1,2</sup>. We report here, a case of expressive aphasia as an isolated residual neurological impairment due to cerebral malaria in a girl child. Full course of the patient was followed and aphasia was reversible in our case.

A 10-yr old girl was admitted in pediatric intensive care unit with complaints of high grade fever with chills for 5 days, altered sensorium, poor feeding for one day and one episode of seizure six hours before admission. Altered sensorium was accompanied with headache and vomiting. Seizure episode lasted for about 2–3 min and was of generalized tonic-clonic type. Past history was uneventful. On examination, Glasgow coma scale (GCS) was : Eye opening (E) 2; Vocalisation (V) 3; Motor responses (M) 3 (total score=8). Blood pressure and heart rate were within normal limits. Patient was febrile (103°F). She was maintaining oxygen saturation at room air. Systemic examination also revealed pallor and splenomegaly (5 cm). In view of repeat seizure, patient was given diazepam intravenous bolus and loaded with injection phenytoin (20 mg/kg) and maintenance doses were charted after 12 h. Empirical treatment on lines of complicated malaria was started as per WHO guidelines<sup>3</sup>. Blood investigations revealed Hb 4.6 g%, TLC 4600, DLC: P-64 L-30 E-5 M-1, Platelet count 16000/mm<sup>3</sup>. Peripheral blood smear examination showed ring forms of *Plasmodium falciparum*. Blood sugar, renal function tests (including electrolytes) and liver function tests were within normal limits. Keeping in view the clinical findings and laboratory reports, diagnosis of cerebral malaria was made and treatment was continued as per WHO guidelines including blood component therapy<sup>3</sup>. Magnetic resonance imaging (MRI) of brain was also normal. Over the course of treatment, after 3 days, the child improved with only exception of not being able to speak. She was fully alert and was able to comprehend verbal commands and she could indicate things by signs.

Clinical examination, hearing tests, detailed ear, nose and throat examination did not suggest any organ abnormality. To find the cause of expressive aphasia, repeat MRI brain was done on Day 10 of admission and it was normal. Keeping the clinical course in mind, the diagnosis of post-cerebral malaria expressive aphasia was made. Speech therapy was given daily in hospital. On Day 16 of admission the child started speaking meaningful words and after 3 following days, the child was having normal speech. After 19 days of admission, child was discharged and on follow-up she is doing well.

## DISCUSSION

Cerebral malaria is the most dreaded complication of malarial infection. Even with modern adequate medical treatment, the survivors of cerebral malaria do have residual neurological sequelae. In one study on neurological deficits following cerebral malaria in Indian adults, authors found various neurological sequelae including aphasia<sup>4</sup>. In available literature, the various neurological deficits including speech deficits have been reported to be transient in nature<sup>4,5</sup>. In other studies, however, workers found speech deficits to be significantly more and persisting in cerebral malaria survivors<sup>6,7</sup>. In our patient, the aphasia was regularly being managed with the help of a speech therapist. It resolved in about two weeks and thus it was purely reversible in nature.

The mechanism behind the aphasia and other neuropsychiatric deficits in cerebral malaria is not exactly known. Observational studies have identified repeated and prolonged seizures, intracranial hypertension, severe metabolic derangement, deep and prolonged coma as risk factors for poor outcome<sup>8</sup>. There was no abnormality on MRI brain in our case. Thus, neurotoxic/immunological mechanisms (of ultrastructural microscopic/biochemical nature) may be involved in our case as cause of expressive aphasia.

There is paucity of literature on post-cerebral malaria expressive aphasia in children from Indian subcontinent. Neurological deficits are known to be higher in children than adults, in cerebral malaria<sup>1,2</sup>. There is a need to create Indian epidemiological database of aphasia and other neurological deficits due to cerebral malaria, especially in children (in whom these occur more). In areas of poor medical healthcare facilities, we can predict a large number of speech deficits in patients secondary to cerebral malaria. Speech deficits are also a significant cause of secondary cognitive and behaviour dysfunction. Carter *et al*<sup>6</sup> laid big emphasis to provide good rehabilitation services in resource poor societies, side by side to preventive and curative measures, to improve the outcome. This is especially true in the light of the fact that being resource poor, areas of high malaria transmission are poor in rehabilitation facilities too. Scientific studies can help us in directing our rehabilitation efforts to only those deficits which are long-lasting or permanent. Unnecessary investigations and treatments can be avoided for complications that are transient/reversible in nature.

Present case highlights some important facts:

1. Expressive aphasia isolatedly can present as residual deficit after recovery in *P. falciparum* cerebral malaria; and.
2. If properly cared and followed up, it may recover fully. To our knowledge there is scant literature from Indian subcontinent about this complication and its full

course. We followed this case fully and the aphasia reverted in two weeks time. Whether post-malarial aphasia is actually transient in its natural course, or improves by good rehabilitation (as in our case), should be subjected to further scientific exploration, by doing large follow up studies.

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Correspondence to: Dr Baljeet Maini, Assistant Professor, Department of Pediatrics, Maharishi Markandeshwar Institute of Medical Sciences and Research (MMIMSR), Mullana, Ambala–133 207, India.  
E-mail: b\_maini@rediffmail.com

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