Cerebellar Atrophy in Falciparum Malaria

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Severe neurological complications are associated with falciparum malaria. We describe the case of an eight-year-old male child with severe falciparum malaria with high-level parasitemia and severe thrombocytopenia. There were features of abnormal gait, speech difficulty and altered behavior pattern during the recovery phase. This occurred even after receiving antimalarial therapy. MRI showed bilateral cerebellar atrophy.

Key Words: *Cerebellar atrophy, Complications, Plasmodium falciparum.*

We report a case of falciparum malaria which showed features of neurological sequelae in the form of persistant cerebellar signs, abnormal gait with speech difficulty and abnormal behavior even after receiving antimalarial therapy. On follow up, child's cerebellar signs persisted and MRI showed features of cerebellar atrophy.

CASE REPORT

An 8-year-old male child from a malaria endemic area was admitted to our hospital with high grade fever, generalized tonic clonic seizures with altered sensorium for a period of two days. He was born of non consanguineous marriage with normal birth history, normal development and was fully immunized. There was no associated history of measles, febrile seizures, allergy, anaphylaxis or contact with tuberculosis. On examination, there was altered sensorium, severe pallor, icterus with hepato-splenomegaly. Child was provisionally diagnosed as cerebral malaria and was treated with artesunate, doxycycline and ceftriaxone.

Investigations revealed hemoglobin of 4.7g/dL, total leukocyte count of 8,800 mm^3, differential count showed $N_{60}\ L_{38}E_0\ M_2$, platelet count 30,000 mm^3 and peripheral smear showed ring forms and gametocytes of plasmodium falciparum with high-level parasitemia. Liver enzymes were raised with a serum bilirubin of 6 mg/dL. Serum values of glucose, urea, calcium and electrolytes were normal. The chest *X*-ray, CSF study and CT brain were normal.

Blood transfusion was given to correct anemia. The child's sensorium and general condition improved on the 6^{th} day of hospitalization. Child was discharged after ten days of hospital stay and advised regular follow-up. The neurological status of the child was normal at the time of discharge.

Child was readmitted to our hospital three months later with features of unsteady gait, slurred speech, clumsy hand movements and abnormal behavior. Child was conscious, hyperalert, and occasionally obeyed commands. He had difficulty in maintaining balance while walking and there was truncal ataxia with bilateral cerebellar signs. There was no neck rigidity. Cranial nerves were normal. On motor examination, he had normal tone with power 4/5 in all limbs. Sensory system examination was unremarkable. Bilateral plantar reflex was extensor. Examination of ears and eyes were normal.

The routine hematological and biochemical investigations were normal. EEG study and CSF study for measles antibody was negative. In view of the neurological complications MRI was done. MRI images showed diffuse atrophy of vermis and both cerebellar hemispheres. The subarachnoid space in the posterior fossa and cisterna magna were dilated. Cerebral cortex, brain stem, thalamus and basal ganglia were normal. Child was advised physiotherapy with special emphasis on pelvic muscle exercises and gait training. Parents were counseled regarding the condition of the child and were adviced for regular follow-up.

During the subsequent follow-up, there was improvement in eye-contact and child was consistently obeying commands. The frequency of behavioral problems had also reduced. However ataxia, involuntary movements and speech problems were persisting.

DISCUSSION

Severe neurological complications are associated with

complicated and severe falciparum malaria [2]. Its overall incidence is 0.1% in patients with falciparum malaria [3]. Despite adequate treatment, 10.5% of survivors develop sequelae in the form of psychosis, ataxia, hemiplegia, cortical blindness, aphasia and extrapyramidal signs [4]. Cerebellar involvement in malaria was first reported by Deaderic in 1909 and later on, Ringdon, *et al.* [5] in their pathological study demonstrated a definite involvement of cerebellum in patients who died of cerebral malaria as well as in experimental animals. Cerebellar involvement in falciparum malaria can occur during the acute stage of fever or as a sequelae of cerebral malaria in survivors. Two syndromes of cerebellar ataxia have been recognized, acute and delayed cerebellar ataxia [6]. A syndrome of delayed cerebellar ataxia after afebrile phase of a few days to weeks after an attack of fever attributable to plasmodium falciparum has been reported from Srilanka, India and Africa. Senanayake, *et al.* [7] reported the clinical features of delayed cerebellar ataxia following falciparum malaria.

Some children experience a post-malaria neurological syndrome after recovery from a complicated falciparum infection [8]. Nguyen, *et al*. [9] in 1996 described a post malaria neurological syndrome in 1.8% of the patients after severe falciparum infections in Vietnam. It was described as the presence of neurological or psychiatric symptoms within two months after recovery of acute infection with malaria. However, till date there are probably no definitive reports of the occurrence of cerebellar atrophy in falciparum malaria after receiving antimalarial therapy. Most of reports suggest recovery from episodes of cerebellar syndrome.

The pathogenesis of cerebellar syndrome is due to the obstruction of micro-circulation due to sludging of parasitized RBCs and direct malarial vasculopathy, causing extensive damage to Purkinje cells of the cerebellum. The purkinje cells are also susceptible to damage due to hyperpyrexia. Immunopathological events include cytoadherence, release of cytokines like TNF-á, IL-2 and IL-6, vascular leakage, edema and tissue anoxia in the brain. Severe gait and truncal ataxia are striking features suggesting that the disease predominantly affects midline cerebellar structures and the majority of patients are afebrile before the onset of cerebellar symptoms [10].

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