Case Report

Falciparum Malaria with Multiple Neurological Complications – A Case Report

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ABSTRACT

Malaria remains the common cause of childhood infections. A 2 year-old female child from a malaria endemic area was admitted to our hospital with high grade fever for 3 days, multiple episodes of generalized tonic clonic seizures (GTCS) for 1 day and altered sensorium on day 4. We report a case of falciparum malaria in a 2 year old girl presenting with seizures and other neurological deficits like hemiplegic, aphasia and cortical blindness.

Keywords:

Falciparum malaria, neurological complications, plasmodium falciparum (PF) hemiplegia

INTRODUCTION

Malaria is one of the most common infectious childhood illness, affecting more than 300-500 million reported children globally every year, of which more than one million cases result in death. Malaria parasites infect about 650 million people worldwide and P. falciparum alone leads to almost one million deaths per year making it the most virulent parasite causing malaria. It is pertinent to develop efficient means of controlling plasmodium falciparum (PF) in areas to which malaria infections are highly endemic. The most severe complications of the disease are primarily due to infection with plasmodium falciparum (PF).

Most malarial deaths occur among infants and young children.[1] Four species of plasmodium, P. vivax, P. falciparum, P. ovale and P. malariae cause nearly all the malarial infections in humans. Of the four species, falciparum is the most dangerous, since it causes more severe manifestations which culminate in multi system failure. Several neurological complications are associated with complicated and severe falciparum malaria, which is rarer than other forms of malaria.[2] The common central nervous complications of acute malaria are febrile convulsions and cerebral malaria.

We report a case of falciparum malaria in a 2 year old girl presenting with seizures and other neurological deficits like hemiplegic, aphasia and cortical blindness.

CASE REPORT

A 2 year-old female child from a malaria endemic area was admitted to our hospital with high grade fever for 3 days, multiple episodes of generalized tonic clonic seizures (GTCS) for 1 day and altered sensorium on day 4. She was born of non-consanguineous marriage with normal birth history, normal development and was fully immunized. There was no past history of measles, febrile

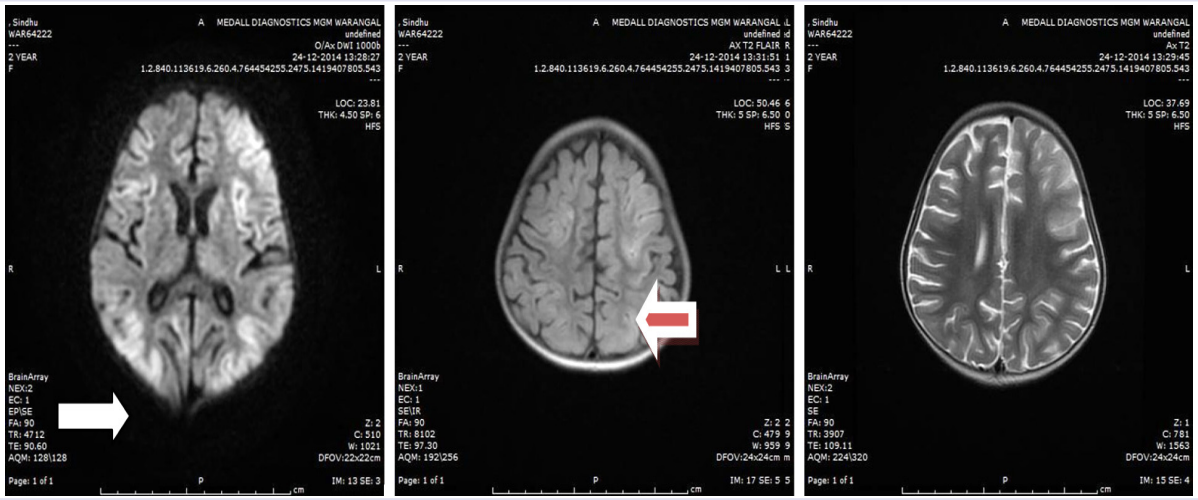

Figure 1 : Diffuse asymmetric edema involving bilateral cerebral hemispheres without diffussion restriction without hemorrhage

seizures, allergy, anaphylaxis or contact with tuberculosis. No previous hospital admissions.

On examination, child was in altered sensorium with Glascow coma scale (GCS) E2V1M4, severe pallor and hepato splenomegaly. Central nervous System examination revealed normal pupils with right hemiparesis, and cranial nerves were intact. Rapid diagnostic tests for (RDT) for PV/PF showed PF positive. Child was provisionally diagnosed as cerebral malaria and was treated with artesunate, ampicillin, ceftriaxone and clindamycin.

A single dose of primaquine was given on 7th day of admission. Investigations revealed hemoglobin of 4.0 g/dL, total leukocyte count of 12,400/mm3, platelet count 87,000/mm3 and peripheral smear showed microcytic hypochromic anemia with neutrophilic leucocytosis. Smear for malarial parasite was positive for falciparum. Liver enzymes were normal. Serum creatinine, blood urea, and ABG were normal. Serum values of glucose, calcium and electrolytes were normal. The chest X-ray and CSF study were normal.

Her vitals were found to be normal. On 2nd day of admission her GCS was E4V1M4 and developed absent menace reflex (Cortical Blindness). Tone was decreased in right upper and lower limb with power 2/5 child eventually developed aphasia.

Plain CT scan showed hypodense lesion left frontal lobe s/o edema. CECT showed multiple hypodense lesions in the bilateral parietal temporal and frontal lobes s/o multiple infarcts. Child was treated with mannitol to relieve cerebral edema. Blood transfusion was given to correct anemia. MR angiography and venography showed diffuse asymmetric cortical edema with no diffusion restriction noted involving b/l cerebral hemispheres. Physiotherapy was advised, the child's sensorium and general condition was gradually improved. Child was discharged after fifteen days of hospital stay child was conscious, coherent and has recovered from hemi paresis prescribed iron supplements, high protein diet and advised regular follow-up. However aphasia and cortical blindness were persisting.

DISCUSSION

Severe neurological complications are associated with complicated and severe falciparum malaria.[3] Its overall incidence is 0.1% in patients with falciparum malaria.[4] Despite adequate treatment, 10.5% of survivors develop sequelae in the form of psychosis, ataxia, hemiplegia, cortical blindness, aphasia and extrapyramidal signs.[5] Case fatality rate was 13.3% 95% CI (11).

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.[6] In available literature, the various neurological deficits including speech deficits have been reported to be transient in nature.[6, 7]

The mechanism behind the aphasia and other neuropsychiatric deficits in cerebral malaria is not exactly known. 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. [8, 9] 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). One study of cortical blindness in a tertiary ophthalmic clinic in Nigeria found eight of 22 (36%) cases were caused by cerebral malaria. [10]

We infer that lack of visual behaviour acutely after cerebral malaria is not due to retinopathy, and that it is usually associated with other sensory or motor deficits. It indeed appears most likely to be a cortical phenomenon. In cerebral malaria intravascular sequestration of erythrocytes parasitized by P falciparum occurs in both the brain and the retina.

The parasites metabolise oxygen and haemoglobin, which we believe causes a relative deoxygenation or hypoxia leading to intracellular oedema, and hence reversible opacification with resolution of the metabolic deficit intracellular oedema would reverse, allowing a normal visual acuity on recovery.

CONCLUSION

CNS complications of malaria should be suspected in patients with malaria endemic areas presenting with neurological symptoms. Early antimalarial therapy can lead to dramatic improvement of neurological manifestations, as occurred in our case recovery from hemiparesis.

CONFLICT OF INTEREST

The authors declared no conflict of interest.

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