

Case Report

Cerebellar Malaria Due to *Plasmodium vivax* in a Child

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Abstract

Infection resulting from *Plasmodium vivax* is the commonest type found in India as well as other tropical regions. The clinical picture is variable depending on the nature, immunity of the child, and lives in an endemic area or not. Here, we report a child, who presented with cerebellar dysfunction with intact sensorium and *Plasmodium vivax* was positive on peripheral smear.

Keyword: Plasmodium vivax, Cerebellar, Malaria, India

Introduction

Malaria is an acute or chronic illness characterized by paroxysms of fever, chills, sweats, fatigue, anemia and splenomegaly. Cerebellar ataxias, extrapyramidal rigidity and cranial nerve palsies presenting with intact sensorium are rare focal neurological deficits reported in malaria (1). Here, we report a child, who presented with cerebellar dysfunction with intact sensorium; *Plasmodium vivax* was present on peripheral blood smear and responded very well to antimalarial treatment.

Case Report

A 10-year-old male child from a malaria endemic area was admitted with high grade, intermittent type of fever since 3 days earlier. The child had difficulty in walking, unsteadiness of gait and tremors from the Second day of illness. He had no history of convulsion, unconsciousness, & bladder or bowel incontinence. On examination, he was fully conscious and well oriented. His vital signs were stable. He had mild pallor with

no icterus or cyanosis. Central nervous system examination revealed ataxia and bilateral cerebellar signs. Cranial nerves were normal. On motor examination, he had normal tone with grade 5 power. Sensory system examination was unremarkable. Bilateral planter reflex was flexor. Per abdominal examination shows mild splenomegaly. Fundus was normal and no sign of meningeal irritation was seen. On investigation, hemoglobin: 9gm/dl, TLC: 6,000/cumm (52% polymorphs, 45% lymphocytes, 2% eosinophils, 1% monocytes), peripheral blood smear shows normocytic and normochromic RBC with ring stage of *P. vivax*. Rapid antigen test (Paracheck HRP-II Antigen detection kit) for *P. falciparum* was negative. Serum urea and electrolyte was normal. No abnormality was detected on chest x-ray. Computed tomography scan of the brain and cerebrospinal fluid examination were normal. The child was put on tab. chloroquine and he responded very well to treatment. Cerebellar signs disappeared by the 3rd day of treatment. On third day, child was put on primaquine tablet was cut for 7 days. After one week on follow-up, there was no fever and cerebellar signs.

Discussion

Malaria is one of the most common infectious diseases and an enormous public-health problem. The disease is caused by protozoan parasites of the genus *Plasmodium*. The most serious forms of the disease are caused by *P. falciparum* and *P. vivax*. Malaria afflicts more than 500 million people, causing from 1.7 million to 2.5 million deaths each year (2). *P. vivax* malaria generally is less severe than *P. falciparum* malaria but may cause death from ruptured spleen as in association with reticulocytosis after anemia. Cerebellar involvement is the most consistent neurological manifestation of complicated as well as of uncomplicated malaria. Purkinje cells are susceptible to damage due to hyperpyrexia. The patients of uncomplicated malaria can also develop cerebellar syndrome (3). Dominant cerebellar involvement could be part of cerebral malaria. As early as 1909, Deaderic reported cerebellar involvement in a case of malaria. Later Ringdon *et al.* reported definite involvement of cerebellum in patients who died of cerebral malaria as well as in experimental animals (3).

The diagnosis of cerebellar malaria can be made based on following features: a) isolated cerebellar symptoms of acute onset associated with fever b) presence of malarial parasite in the peripheral blood c) response to antimalarial drugs. Senanayake *et al* (4) reported the clinical features of delayed cerebellar ataxia following falciparum malaria in patients whereas Koibuchi (5) reported acute disseminated encephalomyelitis following *P. vivax* malaria. The high index of suspicion is rewarding most of the times because the disease has bizarre manifestations especially in young children, septicemia and encephalitis illnesses predominate. Immunopathologic events in patients with malaria include polyclonal activation resulting in hypergammaglobulinemia and formation of immune complexes, immunodepression, and release of cytokines. Cytoadherence of the infected erythrocyte to vascular endothelium occur in *P. falciparum* malaria (6). It may

lead to obstruction of blood flow and capillary damage with resultant vascular leakage of protein and fluid, edema, and tissue anoxia in the brain, heart, lung, intestine and kidney. Severe gait and truncal ataxia are striking features suggesting that the disease predominantly affects midline cerebellar structures (7, 8). The majority of patients have afebrile period before the onset of cerebellar symptoms. It is always associated with *P. falciparum* infection very rarely reported with *P. vivax* malaria (9).

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