

Cerebellar Malaria: A Rare Manifestation of Plasmodium Vivax

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Citation

B Taksande, U Jajoo, M Jajoo. *Cerebellar Malaria: A Rare Manifestation of Plasmodium Vivax*. The Internet Journal of Neurology. 2006 Volume 7 Number 1.

Abstract

Sir, P. vivax malaria is acute and excruciating, involving repeated episodes of high fever preceded by violent headache and chills and profuse sweating, and often accompanied by vomiting, diarrhea, and enlargement of the spleen (1). Cerebellar ataxias, extrapyramidal rigidity and cranial nerve palsies presenting with intact sensorium are rare focal neurological deficits reported in malaria (2). Here we report a case of Plasmodium Vivax malaria, who presented with unilateral cerebellar dysfunction with intact sensorium and responded well to antimalarial treatment.

A 50 yr. old man from a malaria endemic area presented with fever and headache for 2 days. The relatives noticed imbalance and swaying on the right side, on the 2nd day of illness. He had no history of seizures, loss of consciousness or bladder or bowel incontinence. On admission, he was oriented and obeying commands (Glasgow coma score 14/15). His vital signs were stable and he had no icterus or neck stiffness. Cranial nerves were normal. On motor examination, he had normal tone with grade 5 power. He had right sided cerebellar signs and ataxia. He had an bilateral plantar flexor response. Sensory system examination was unremarkable. Fundus was normal. Routine biochemical and hematological examination was normal. Peripheral blood showed the evidence of a ring stages of P. Vivax. Magnetic resonance imaging (MRI) of the brain and cerebrospinal fluid (CSF) examination was normal. He was treated with chloroquine injection and IVF. His fever responded and cerebellar signs on the right side improved completely by the 3rd day of treatment. At 2 weeks follow-up, there were no cerebellar signs.

Plasmodium vivax accounts for approximately 70-80 million cases annually (or 20-percent of the global burden of 350-500 million total cases of all human malarial). 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. Cerebellar signs resolve along with cerebral manifestations (2). Senanayake N et al (4) reported that the clinical features of delayed cerebellar ataxia following falciparum malaria in patients. This unusual complication has an acute onset, with signs suggesting a predominantly right sided cerebellar lesion without any evidence of cerebral involvement. Whereas Koibuchi T (5) reported that acute disseminated encephalomyelitis following plasmodium vivax malaria from the clinical course and MR images in a 24 year old Japanese men. Severe gait and truncal ataxia are striking features suggesting that the disease predominantly affects midline cerebellar structures. The majority of patients have afebrile period before the onset of cerebellar symptoms. It is always associated with Plasmodium falciparum infection. The exact mechanism of delayed cerebellar ataxia is unknown, however, there is some evidence to suggest involvement of immunological factors in the pathogenesis (6). The pathogenesis of cerebellar malaria is the same as that of cerebral malaria, being caused by the obstruction of microcirculation by the sludging of parasitized RBC and a direct malarial vasculopathy resulting in increased vascular permeability. There is extensive damage to the Purkinje cells of the cerebellar cortex associated with hemorrhage, small infarctions and microglial infiltration. But these changes are not specific for malaria.

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