

DELAYED CEREBELLAR ATAXIA COMPLICATING FALCIPARUM MALARIA

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A wide variety of neurologic abnormalities have been described in association with falciparum malaria, the most serious of which is cerebral malaria.¹ Delayed cerebellar ataxia is an unusual complication of falciparum malaria.² Most of the cases have been reported from Sri Lanka, and a few from India and Africa.²⁻¹⁰ It is characterized by an acute onset, with signs suggesting a predominantly midline cerebellar lesion without any cerebral involvement. Delayed cerebellar ataxia occurs about 13 days after the onset of an attack of otherwise uncomplicated falciparum malaria. It is self-limiting, with spontaneous and complete recovery within three months.² We recently saw a patient who developed this complication of falciparum malaria following a visit to Africa. The case is described here and the pertinent literature reviewed.

Case Report

A 24-year-old Qatari male patient was admitted to Hamad General Hospital in December 1997 with a complaint of fever, headache, dizziness, and vomiting, a few days after returning from a visit to the Comoros Islands. The patient recalled taking medicine for malaria prophylaxis during his stay there, but did not know what type. He consulted a physician in the army clinic, whose laboratory investigations showed hemoglobin level of 14.5 g/dL, WBC 2300/mm³ and platelets of 112,000/mm³. The blood film for malaria revealed ring forms, however, the species was not specified. Bilirubin was 26 µmol/L, alkaline phosphatase 216 U/L, alanine aminotransferase (ALT) 95 U/L, aspartate aminotransferase (AST) 58 U/L, and serum creatinine 109 µmol/L. The patient was given three tablets of Fansidar as a single dose. Two days later, he was still highly febrile. Physical examination revealed temperature of 40°C, blood pressure of 110/70 mm Hg, pulse 110/minute, and palpable spleen, otherwise physical examination was normal. Repeat investigation revealed hemoglobin to be 14.5 g/dL, WBC 3900/mm³, platelets 40,000/mm³, negative blood film for malaria, and negative

blood culture. He was given quinine sulphate 600 mg three times daily for three days, and doxycycline 100 mg twice for a presumptive diagnosis of falciparum malaria. He tolerated the medication well, with no side effects, specifically hypoglycemia. He showed good response and became afebrile. Two days after stopping medication, he started to complain of dizziness and instability of gait. His physical examination was unremarkable apart from ataxia of central truncal type, dysarthria, abnormal finger-nose test, heel-shin test, and repetitive fine movements. Investigations revealed Hb 13.6 g/dL, platelets 211,000/mm³, WBC 3900/mm³, ALT 81 U/L, AST 25 U/L, bilirubin 14 µmol/L, alkaline phosphatase 150 U/L, and negative blood film for malaria. Renal function tests and electrolytes were normal. Computed tomographic scan of head was normal. Because of persistent ataxia, the patient was transferred to a hospital in the United Kingdom for further neurologic investigation. Repeat blood film for malaria was negative, but a parasite-F test confirmed the diagnosis of recent *Plasmodium falciparum* infection that had been effectively treated. Magnetic resonance image of head, and electroencephalogram were normal. Cerebrospinal fluid (CSF) examination was normal, and polymerase chain reaction studies for varicella and Epstein-Barr virus in CSF were negative. The patient was diagnosed as a case of delayed cerebellar ataxia complicating falciparum malaria. He received symptomatic treatment in the form of prochlorperazine, with gradual improvement of his condition and complete recovery over a two-month period.

Discussion

Delayed cerebellar ataxia is an unusual complication of falciparum malaria. It was first reported by Senanayake et al. in 1984.¹¹ Most of the previous cases have been reported from Sri Lanka, with a few from India and Africa. The disease occurs in an otherwise healthy conscious patient following an attack of uncomplicated malaria. The period between the fever and ataxia varies from 3 to 41 days (mean 13 days).² The ataxia is mainly truncal, suggesting a midline cerebellar lesion. Patients are usually conscious, rational, and alert and have no signs of cerebral involvement, in contrast to those with cerebral malaria. Peripheral blood film may show gametocytes, schizonts of

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Plasmodium falciparum, or may be negative for malaria in some patients. Laboratory investigations in these patients, including CBC, CSF examination, electroencephalography, and CT scan of the brain, are usually normal.² Diagnosis is made after exclusion of other causes of ataxia such as drugs, toxins, alcohol abuse, and viral causes such as varicella, rubella, and rubeola.⁸ Etiology is not known, but an immunologic mechanism has been suggested to play a role in the pathogenesis of the condition.¹² De Silva et al. suggested that the absence of cases before 1984, and the fact that most cases were reported from Sri Lanka, could be explained by the appearance of a new strain of *Plasmodium falciparum* in that area.¹³ The disease is self-limiting with excellent prognosis, and complete recovery occurs over a period of three months.² Treatment is only symptomatic, although steroids have been used in some patients with good response.⁸ This patient is the first case of delayed cerebellar ataxia following falciparum malaria that was seen in our hospital over the last 15 years, in spite of the large number of cases of falciparum malaria we see in patients from the Indian subcontinent and Africa.

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