Case Report

Delayed cerebellar ataxia: A rare self limiting complication of plasmodium falciparum malaria

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Abstract

The classic presentation of malaria with paroxysms of fever is seen only in 50-70% of the patients. The development of immunity, the increasing resistance to anti-malarial drugs, and the indiscriminate use of anti-malarial drugs have led to malaria with the presentation of unusual features. Cerebellar ataxia, extrapyramidal rigidity and various psychiatric symptoms have been described either as early manifestations of cerebral malaria or as a part of post malaria neurological syndrome. In this case report, we will discuss one such patient of falciparum malaria infection who developed midline cerebellar signs, and responded to anti-malarial treatment.

Key Words: Delayed cerebellar ataxia, neurological complications of plasmodium falciparum malaria, plasmodium falciparum malaria

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INTRODUCTION

Way back from 1882, intensive efforts are being made to understand malaria and methods to control it. Malaria is a major public health problem in the developing world owing to its high rates of morbidity and mortality. An estimated 300-500 million people contract malaria each year, resulting in 1.5-2.7 million deaths annually. Of all the malarial parasites that infect humans, Plasmodium falciparum is most commonly associated with neurological complications. The neurological complications of falciparum malaria are common and encompass a wide spectrum of clinical presentation. These complications can manifest during acute illness, or can present during convalescence. Increasing drug resistance[1] in several parts of our country has further aggravated the problem of management. Delayed cerebellar ataxia is an unusual complication of falciparum malaria. It was first reported by Senanayake *et al*. in 1984. This complication is predominantly found in Sri Lanka though a few cases have been reported from India and Africa.

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CASE REPORT

A 23-year-old pre-morbid healthy male patient was referred to our hospital with complaints of giddiness and loss of balance while walking since 1 month associated with mild, throbbing, generalized headache. There was no history of nausea, vomiting, seizure, syncope, preceding head injury or any addictions. About 1 month back, patient was admitted to a local hospital

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with complaints of fever with chills, for which patient was treated with Ceftriaxone and Amikacin for 3 days. No history of patient having received anti-malarial in any form. Patient's fever did subside but then he developed giddiness and started requiring support for walking. Patient received symptomatic treatment but had no symptom relief hence a CT scan of head was done, which was apparently normal. There was no drug-history suggestive of cause for his ataxia. Due to persistence of symptoms the patient was referred to our center for further management. On admission to our hospital, his pulse was 72/min, blood pressure 100/70 mm of Hg, respiratory rate 18 cycles/min, afebrile, pallor was present; no icterus, cyanosis, clubbing, edema; jugular venous pressure was normal. There was no evidence of any neurocutaneous markers. Cardiovascular and respiratory systems' examination were unremarkable. Per abdomen examination was suggestive of splenomegaly (palpable 2 cm below the left costal margin), no hepatomegaly. On neurological examination, he was fully alert, conscious and oriented to time, place and person. On higher mental examination only attention span was reduced. All cranial nerves and sensory system were normal. On motor system examination, he had hypotonia in all limbs. All superficial reflexes were normal with bilateral plantars flexors. Deep tendon reflexes were normal in upper limbs and exaggerated in lower limbs with pendular knee jerks in both the lower limbs. Finger-Nose Test, Knee-Heel test, Dysdiadokokinesis, Tandem Gait were bilaterally impaired. Nystagmus-Absent. There were no signs of meningeal irritation. His laboratory investigations were as follows: Hb-9.9 G%, WBC-5800 cumm of blood, Polymorphs-70% Lymphocytes-26%, Monocytes-1%, Eosinophils-3%, Platelet Count-1.8 lakh. Erythrocyte sedimentation rate 110 mm at the end of 1 h.

Peripheral smear showed gametes and rings of plasmodium falciparum malaria with parasitic index-2.8%. Random blood sugar-83 mg/dl, blood urea-22 mg/dl, serum creatinine-1.2 mg/dl, serum sodium-136 mEq/L, serum potassium-3.8 mEq/L.

MRI Brain-Normal study.
Cerebrospinal Fluid Analysis-Normal study.
Serum Vitamin B12 levels-Within normal limits.
Serum Thiamine levels-Within normal limits.
Chest X-Ray PA View-Within normal limits.
ECG 12 Leads-Within normal limits.
USG (Abdomen + Pelvis)-Splenomegaly (16 cms).

He was started on quinine sulphate orally, along with pyrimethamine and sulfadoxine on doses recommended by WHO 2010 guidelines for treatment of malaria. His cerebellar signs showed a complete recovery in 14 days with waxing and waning phenomenon. He did not require any steroids. He was discharged after 14 days in a hemodynamically stable condition and with no neurodeficit.

DISCUSSION

Delayed cerebellar ataxia is one of the unusual complications of falciparum malaria. Cerebellar involvement in plasmodium falciparum malaria can occur in:

1. Acute stage of fever,
2. In survivors of patient with cerebral malaria as resolving stage after full control of infection,
3. In the form of delayed cerebellar ataxia after afebrile latency,
4. As a complication of anti-malarial drugs with CNS toxicity.

It is generally seen in 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] Severe gait and truncal ataxia are striking features, which suggest that the disease has predominantly affected the midline cerebellar structures.[2] Peripheral blood film may show gametocytes, schizonts of 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] Cerebellar involvement appears to be the most consistent neurological manifestation of complicated, as well as of uncomplicated malaria. So far the etiology is not known but a few theories have been postulated-

1. An immunologic mechanism has been suggested to play a role in the pathogenesis of the condition.[3]
2. 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.[4]
3. The Purkinje cells are susceptible to damage due to hyperpyrexia[5,6] causing cerebellar manifestations.

This condition is mostly self-limiting with good prognosis requiring only antimalarials and symptomatic treatment with a few cases requiring steroids.[7]

Thus, the treating physicians practicing in endemic areas of malaria should include neurological complications of P. falciparum malaria as a differential diagnosis in all patients with cerebellar ataxia. Despite cases of neurological manifestations of

P. falciparum malaria being reported since long and research by Senanayake *et al*.,[2] De Silva *et al*.,[3] Garg *et al*.,[5] Kochar *et al*.,[8,9] WHO has not included the various presentations of neurological complications of P. falciparum malaria other than cerebral malaria in the recently issued (2010) guidelines, which means that proper study of understanding all neurological manifestations of P. falciparum malaria and its treatment protocol needs to be done and revised, to help the treating physician identify and treat the disease and its complications at the earliest in order to improve the outcome of the disease.

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