

An Interesting Case Report on Cerebellar and Psychiatric Manifestations in Malaria

MOHAMMED ASHRAF, MADHU KUMAR, SMITHA C. SALDANHA, VEERENDRA K.H

ABSTRACT

Malaria remains a major cause of morbidity and mortality worldwide. 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 present a patient with mixed malaria, who developed bilateral cerebellar signs and a delayed onset of psychiatric manifestations, who responded to anti-malarial treatment.

Key Words: Malaria, Ataxia, Psychosis

INTRODUCTION

Despite intensive efforts over the last century to understand and control malaria, it remains a leading cause of morbidity and mortality in humans. An estimated 300–500 million people contract malaria each year, resulting in 1.5–2.7 million deaths annually. The classic presentation of malaria with paroxysms of fever is seen only in 50%–70% of the patients. In endemic regions, malaria can present with unusual features due to the development of immunity, the increasing resistance to anti-malarial drugs and the indiscriminate use of antimalarial drugs. As a result of lack of awareness of the atypical manifestations, it is not uncommon for malaria to be diagnosed late or even remain unrecognized, resulting in severe illness or death [1]. Cerebral malaria is the most dreaded and a potentially life-threatening complication. Cerebellar ataxia, extrapyramidal rigidity and various psychiatric symptoms have been described either as the early manifestations of cerebral malaria or as a part of the post malaria neurological syndrome [2]. Here, we are describing one such interesting case of malaria with atypical manifestations.

CASE REPORT

A 26 year old non-alcoholic man from a malaria endemic area presented with symptoms of high grade fever, bifrontal headache and four episodes of non-projectile, non-bilious vomiting since 3 days. His relatives noticed slurring of speech, clumsiness of hand movements and unsteadiness of gait on the third day of illness. However, there was no history suggestive of seizures, loss of consciousness, altered sensorium, bowel and bladder incontinence.

At the time of admission, he was conscious and well oriented and obeyed verbal commands. His vital signs were stable and he had no pallor, icterus or neck stiffness. His cranial nerves and fundi were normal. On motor examination, he was found to have a normal tone and power in all the four limbs with flexor plantar response bilaterally. He had bilateral cerebellar signs namely, past pointing, intention tremors and dysdiadochokinesia. There was horizontal nystagmus and scanning speech. The sensory system

examination was unremarkable. Other systems did not reveal any abnormality on examination.

The routine biochemical and haematological investigations provided normal reports. His peripheral blood smear revealed ring forms and gametocytes of *Plasmodium vivax* and *Plasmodium falciparum*. MRI brain with Gadolinium was done to rule out the structural, ischaemic or auto-immune de-myelinating disease, but the study proved to be normal. His CSF analysis revealed no abnormality. His vasculitic work up was normal.

The patient was managed as a case of cerebral malaria with intravenous quinine and antibiotics (third generation cephalosporins). His fever touched the baseline within a week and the cerebellar signs subsided after 10 days of hospital stay. However, he became irritable and agitated and had waxing and waning of mood and a decreased attention span. The patient had auditory and visual hallucinations and described himself as free and as “flying as a *pari* (angel)”. He also showed signs of social disinhibition. A review of his past history disclosed no evidence of psychiatric illness in him or in his family. He was started on a low dose of Quetiapine and was given sedatives at night. He recovered completely in a span of two weeks.

DISCUSSION

Several neurological complications are associated with complicated and severe falciparum malaria. A large number of survivors are left with disabling neurological sequelae. Few patients may experience the post-malaria neurological syndrome after recovery from a complicated falciparum infection [2]. Various psychiatric syndromes have been described either as an early manifestation of cerebral malaria or as a part of post malaria neurological syndrome [2-3]. In India, several cases of delayed cerebellar ataxia have been described following recovery from clinical malaria [4-6].

Despite adequate treatment, 10% to 18% of the survivors develop neurological sequelae in the form of psychosis, ataxia, hemiplegia, cortical blindness, aphasia and extrapyramidal syndrome. In a large series of patients from India, including 185 adult patients,

Bajija and Kochar observed neurological sequelae in 13 (10.5%) of the 123 survivors. These neurological sequelae were in the form of psychosis, cerebellar ataxia, extrapyramidal rigidity and hemiplegia. All of them recovered fully at the end of the month [4].

Malarial psychosis develops because of encephalopathy in patients with cerebral malaria. It manifests as paranoid and manic syndromes in the acute stage, depression being a late sequelae. Agitation and confusion may develop after the patient has recovered from coma. These psychiatric manifestations may be the presenting features in patients with acute uncomplicated malaria, especially in association with hyperpyrexia [2]. Neuropsychiatric manifestations are also caused by antimalarial drugs like mefloquine, which was not used in our patient.

Cerebellar involvement is the most consistent neurological manifestation of complicated as well as of uncomplicated malaria. The Purkinje cells are susceptible to damage due to hyperpyrexia [2, 7]. A syndrome of delayed cerebellar ataxia has frequently been reported from Sri Lanka, in which there was an acute, self limiting, isolated ataxia without any evidence of cerebral involvement. Severe gait and truncal ataxia are striking features which suggest that the disease has predominantly affected the midline cerebellar structures [8]. A prospective study conducted in Vietnam, described 22 patients including 19 adults and 3 children, who had neurological or psychiatric symptoms which occurred within 2 months after acute and cured malarial manifestations. Among 18,124 patients with treated *P. falciparum* malaria: overall, the incidence was 0.7 to 1.8 per 1000 people [9].

Our case suggests that malaria may have many presentations. A broader definition of the neurological complications and the associated signs and symptoms of the disease may help in understanding the pathobiology of the syndrome and it could also shed some light on the underlying mechanisms.

REFERENCES

- [1] SA, Shanbag P. Atypical Manifestations of Malaria-Review. *Research and Reports in Tropical Medicine* 2011; 2: 9-22
- [2] Garg RK, Karak B, Misra S. Neurological manifestations of malaria: an update. *Neurol India* 1999;47:85-91
- [3] Blocker WW, Kastl AJ, Daroff RB : The psychiatric manifestations of cerebral malaria. *Am J Psychiatry* 1968; 125 : 192-96.
- [4] Kochar DK, Kumawat BL, Kochar SK, Bajija HN, Maurya RK. Delayed cerebellar ataxia, a complication of *Plasmodium falciparum* malaria. *J Assoc Physicians India* 1996; 44: 686-88.
- [5] Kochar DK, Shubhakaran, Kumawat BL, Kochar SK, Halwai M, Makkar RK, et al. Cerebral malaria in Indian adults: a prospective study of 441 patients from Bikaner, north-west India. *J Assoc Physicians India* 2002; 50:234-41.
- [6] Kochar DK, Shubhakaran, Kumawat BL, Thanvi I, Aggrawal N. Cerebellar syndrome in *Plasmodium falciparum* malaria. *QJM* 1999; 92: 233-34.
- [7] Taksande B, Jajoo U, Jajoo M. Cerebellar Malaria: A Rare Manifestation of *Plasmodium Vivax*. *The Internet Journal of Neurology*. 2007;7(1)
- [8] Senanayake N, de Silva HJ. Delayed cerebellar ataxia complicating *falciparum* malaria: a clinical study of 74 patients. *J Neurol*. 1994; 241: 456-59.
- [9] Nguyen TH, Day NP, Ly VC, Waller D, Nguyen HP, Bethell DB, et al. Post-malaria neurological syndrome. *Lancet*. 1993; 348: 917-1001.

AUTHOR(S):

1. Dr. Mohammed Ashraf
2. Dr. Madhu Kumar
3. Dr. Smitha C. Saldanha
4. Dr. Veerendra K.H.

PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of Medicine
Fr Muller Medical College, Mangalore, India.
Ph.: +919448428974; E-mail: drashrafmedd@hotmail.com
2. Post graduate Resident; Department of Medicine
Fr Muller Medical College, Mangalore, India.
Ph.: +919743105248; E-mail: dhuku33@yahoo.co.in.

3. Assistant Professor, Department of Medicine
Fr Muller Medical College, Mangalore, India.
Ph.: +919844685166; E-mail: saldanhasmitha@gmail.com
4. Assistant Professor, Department of Medicine
Fr Muller Medical College, Mangalore, India.
Ph.: 9480484311; E-mail: veerendrakh@gmail.com

DECLARATION ON COMPETING INTERESTS:

No competing Interests.

Date of Submission: **Apr 10, 2011**
Date of Peer Review: **Jun 01, 2011**
Date of Acceptance: **Jun 01, 2011**
Online First: **Jul 03, 2011**
Date of Publishing: **Oct 05, 2011**