Pseudobulbar Paralysis — A Sequelae of Cerebral Malaria

MK Mohapatra*, G Sethy**, SC Mohanty***

Abstract

Demyelination may be a pathogenic mechanism of post-malarial neurological sequelae. It can cause pseudobulbar palsy, which has not been documented earlier. In the present communication we report two cases of pseudobulbar palsy after cerebral malaria with evidence of demyelination.

INTRODUCTION

Neurological sequelae in survivors of cerebral malaria are found in about 3 to 10.5% of cases. These include delayed cerebellar ataxia, psychosis, extrapyramidal rigidity, cranial nerve deficit and hemiplegia. Pseudobulbar palsy as a late complication of malaria with evidence of demyelination has not been reported. Here we report two cases of malaria with pseudobulbar palsy as a sequelae of cerebral malaria.

CASE REPORT

Case 1

RD a 16 year male, was admitted with complaints of inability to swallow food and difficulty in speech for 3 days. He was apparently all right 3 days back when he noticed drooling of saliva from mouth with difficulty in swallowing liquid and subsequently to solid food. It was associated with difficulty in speech, which was slow, indistinct, and monotonous without neat intonation of voice. There were emotional outbursts with spontaneous laughter and cry. There was no history of weakness, blurring of vision and convulsion. He was discharged from the same medical ward after being treated for cerebral malaria with jaundice 2 days before the present episode. The sequence of events was fever for 2 days, loss of consciousness for 2 days, hospitalised for cerebral malaria, and stayed for 6 days. Two days after discharge from the hospital he developed the present set of symptoms and came to the hospital on third day. During the previous admission, peripheral blood smear showed ring forms of \textit{P. falciparum}. Other investigations were, Hb - 8.2 gm/dl, total leukocyte count - 8200/mm$^3$, differential count - N; 70%, L; 20%, E; 10%, random blood glucose - 90 mg/dl, S. bilirubin 0.8 gm/dl, B. urea - 22 mg/dl, S. creatinine - 0.8 mg/dl. Peripheral blood smear as well as ParaSight F test (Becton and Dickinson) for \textit{P. falciparum} was negative. CSF analysis showed clear fluid with normal pressure, protein - 80 mg/dl, sugar - 52 mg/dl. He was diagnosed as a case of post-malarial demyelination similar to acute disseminated encephalomyelitis (ADEM). He was treated with methyl prednisolone and recovered completely after a week.

Case 2

LP a 17 year female admitted with complaint of fever for 4 days and loss of consciousness for 2 days. The patient was apparently all right 5 days back when she started fever with chill and rigour, followed by unconsciousness. General examination revealed a lady with average built and nutrition,
temperature - 104°F, pulse rate - 102 beats/mt., blood pressure - 120/80 mm Hg, respiration rate - 20/mt. The patient was in coma with Glasgow coma scale of 5 without any cranial nerve deficit and bilateral extensor plantar. There were no meningeal signs. Investigation showed Hb - 7.0 gm/dl, total count - 8000/mm³, DC-N; 68%, L;20%, E;10%, M;2%, blood glucose - 90 mg/dl, B. urea - 22 mg/dl, S. creatinine - 0.8 mg/dl, S. bilirubin - 0.8 mg/dl. Cerebrospinal fluid (CSF) was within normal limits. Peripheral smear showed ring forms of *P. falciparum* with the parasite count of 4000/µl.

The patient was diagnosed as a case of cerebral malaria and was treated with intravenous quinine di-hydrochloride with a loading dose of 20 mg/kg body weight followed by maintenance dose of 10 mg/kg slowly in 10% dextrose solution with supportive measures.

She became conscious after 72 hours of therapy with a parasitic clearance time of 48 hours. Nasogastric tube and catheter had been removed and quinine had been administered orally.

On 9th day of hospitalisation, while on oral quinine, she suddenly developed difficulty in deglutition and speech as well as drooling of saliva with emotional outbursts. Examination of cranial nerves revealed involvement of bilateral 7,9,10, and 11th cranial nerves. The power was grade III around all groups of muscle with brisk deep tendon reflexes. Plantar was extensor bilaterally. Repeat lumbar puncture showed raised CSF protein (100 mg/dl) with lymphocytosis. CT scan of brain showed low attenuation. MRI could not be done.

A diagnosis of pseudobulbar palsy as a post-malarial complication (demyelination) was made and managed with methyl prednisolone. She recovered completely after 10 days of steroid therapy.

**Discussion**

The neurological signs of these two cases suggested pseudobulbar palsy. As this stereotype clinical picture developed after recovery from falciparum malaria, it is reasonable to assume a causal relationship between the malarial attack and pseudobulbar palsy. The time interval between the febrile episode (onset of malaria) and the development of pseudobulbar palsy suggests the possibility of post-infectious demyelination. The first patient developed the symptoms on 12th day of fever and MRI showed multiple hyperintense lesions in white matter suggesting demyelination. In the second case, the symptoms developed on 13th day of fever, CSF protein was raised, CT scan showed low attenuation, and the patient responded to steroid. All these favoured a diagnosis of ADEM.

ADEM is an autoimmune inflammatory disease of central nervous system, which is characterised by a definite time interval from the onset of infection to the development of neurological deficit. It is associated with raised protein in CSF, low attenuation in CT scan and diffuse demyelination in MRI.³ It has been complicated with viruses like herpes, measles, rubella, and influenza.³ Recently, *P. falciparum* has been postulated as a causative agent for ADEM.⁴,⁵ Two patients of steroid responsive post-malarial neurological complications characterised by aphasia, myoclonus, and tremor have been reported. In them, pathology similar to ADEM had been postulated.⁴ ADEM as another mechanism of delayed cerebellar ataxia has been described recently.⁵

At present, it is not clear whether *P. falciparum* induces demyelination directly or indirectly by activation of dormant neurotropic viruses. Because it is known that some viruses like Herpes are activated during malaria.³ Hence, more research on this aspect of malaria should be done to explain post-malarial neurological sequelae.

**References**