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ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY FOLLOWING FALCIPARUM MALARIA

General Medicine

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ABSTRACT

An adult male with falciparum malaria infection whose parasites have cleared from the peripheral blood, developed acute onset flaccid paralysis. Clinical examination, imaging and nerve conduction study confirmed acute inflammatory demyelinating polyneuropathy occurring after recovery from malaria.

KEYWORDS

INTRODUCTION

Infection with Plasmodium falciparum is known to cause several neurological complications. The most deleterious is cerebral malaria, carrying a mortality of 10–50% in treated patients.[1] Rarely, patients can experience a neurological syndrome following successful treatment, including delayed onset cerebellar ataxia (DCA),[2–4] acute disseminated encephalomyelitis (ADEM), and acute inflammatory demyelinating polyneuropathy(AIDP).[5–9]

In 1994, a study from Vietnam was the first to describe a distinctly unique post-infectious syndrome, the post-malaria neurological syndrome (PMNS).[10] The paper defined PMNS as:patients with symptomatic malaria infection (initial blood smear positive for asexual forms of the parasite), whose parasites have cleared from the peripheral blood and, in cerebral cases,had recovered consciousness fully,who developed neurological or psychiatric symptoms within 2 months of acute illness.[10]

We are reporting here a case of acute inflammatory demyelinating polyneuropathy(AIDP) following recovery from Plasmodium falciparum malaria.

CASE REPORT

62 yrs old gentleman admitted with sudden onset weakness of both lower limbs . There was no history of involvement of upper limbs, unconsciousness, trauma, abnormal movements, bowel and bladder disturbance, facial weakness, difficulty in vision/speech, recent vaccination. History of falciparum malaria 1 month back which was treated with artemisinin based combination therapy and patient was afebrile after that.

On examination, his pulse was 84/minute, regular; blood pressure 128/78 mm of Hg; respiratory rate 18/minute. There was no pallor, no icterus and no lymphadenopathy. In the examination of central nervous system, the higher functions were normal, cranial nerves were intact, and there was lower motor neuron type of paraparesis. Power was diminished in all muscle groups of lower limbs to (Medical Research Council scale) MRC grade 1, while power in the upper limbs was normal in all muscle groups. There were no fasciculations or wasting. The ankle jerks were absent and all the reflexes of lower limbs were diminished. Reflexes of upper limbs was normal. Plantar response was flexor.

Laboratory investigations showed haemoglobin-12.8 gm/dl, TLC-8,200/mm3, DLC-P76 L23 E1, platelet-1.9 lakh/cmm, blood sugar (F)-85 mg/dl, blood urea-26 mg/dl, serum creatinine-1.1 mg/dl, S. Na+-136 mmol/l, S K+-3.9 mmol/l, malarial parasite blood smear was negative. ECG, fundus examination, and X-ray chest were normal.MRI scan of dorsolumbar spine and screening of other spine was within normal limit.

Nerve conduction studies (NCS) were done which showed prolongation of distal latencies in motor nerves, decrease in compound motor action potential (CMAP) in all nerves, temporal dispersion in all nerves, prolongation of F wave latencies, and conduction blocks in few nerves which was characteristic of acute inflammatory demyelinating polyneuropathy.

Patient developed sudden onset respiratory distress and expired due to type II respiratory failure.

DISCUSSION

The etiology of PMNS remains elusive, but several factors suggest that PMNS may be immunologically mediated. The majority of neurological complications of malaria occur during the cerebral form. Formerly thought to be due to cytoadherence of parasitized red blood cells (PRBCs) in the brain microvasculature,[11] recent research indicates that cerebral malaria likely involves a complex imbalance of pro- and anti-inflammatory cytokines.[12] Clark and colleagues provide compelling evidence that cytoadherence of PRBCs in the brain microvasculature may be a secondary effect of the "cytokine storm" that occurs in severe falciparum infection.[13] Clearance of parasitemia, and latency between recovery and symptom onset make the primary cytoadherence mechanism unlikely in PMNS.

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