

Case Report

Polyneuropathy with autonomic involvement in *Plasmodium falciparum* malaria

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Malaria can cause neuropsychiatric manifestations¹: a sequelae of cerebral malaria, predominant neuropsychiatric signs and symptoms without loss of consciousness during acute stage as a presenting feature and, as post-malaria neurological syndrome (PMNS). Only few case reports of peripheral neuropathy of Landry Guillain-Berre syndrome (GBS) type with and without autonomic involvement have been reported from India^{2,3} and Africa⁴.

Case history

A 36-yr old right handed man presented with history of fever, headache, slowly progressive weakness of all four limbs with syncopal attacks without loss of consciousness of 5–6 days duration.

On examination at the time of admission the patient was conscious, febrile, pulse rate varying between 48 and 70 per min, blood pressure 120/70 mm Hg in supine position with postural fall of 30/14 mm Hg in standing position, and respiratory rate 22 per min regular without significant respiratory embarrassment. On neurological examination higher mental functions speech and cranial nerves were normal except minimal nasal blurring of disc margins bilaterally. Power in upper limbs was 4/5 and in lower limbs 4-/5 (MRC grading), deep tendon reflexes were absent in all the four limbs with bilateral flexor planter response. Sensory and cerebellar examination was normal. There were no signs of meningeal irritation, skull and spine were normal.

On investigation, haemoglobin was 9.5 g%, total leucocyte count 8900 per mm³ with 44% lymphocytes and 54% neutrophils, erythrocyte sedimentation rate was 26 mm in first hour, peripheral blood film showed *Plasmodium falciparum* trophozoites and rings. Blood sugar, liver function tests, kidney function tests and electrolytes were within normal limits. Urine examination was normal including porphobilinogen. Nerve conduction study per-

formed after 3 days of admission was suggestive of demyelinating polyneuropathy. Cerebrospinal fluid opening pressure was raised with normal cell count and sugar with proteins being 64 mg%. Other causes of polyneuropathy like organophosphorous poisoning and exposure to heavy metals were excluded by relevant history and investigations. Family history was negative for similar illness.

Patient was put on injection Arteether 150 mg intramuscularly once daily for 3 consecutive days along with 10 and 25% GDW, and GNS. Fluctuations in pulse rate, and digginess settled down to normal. Oral quinine sulphate 600 mg TDS was started on Day 4 for a total of 7 days along with a single dose of 45 mg primaquine. Patient was discharged on Day 10 with complete recovery.

Among various neuropsychiatric manifestations/sequelae of malaria GBS like polyneuropathy, acute and delayed cerebellar ataxia, periodic paralysis are reported mainly from Indian subcontinent^{1–3}. Exact cause of polyneuropathy is not known but has been attributed to immune mediated capillary damage, toxic oxygen radicals, tumor necrosis factor, parasitic emboli obstructing the vasa nervorum, neurotoxin release, nutritional and metabolic disturbances².

The aim of reporting this case is to make an awareness about this potentially fatal but treatable disease if specific treatment is initiated early, and because of paucity of such cases in literature. Furthermore, it is different from the cases reported from South Africa (Sudan)⁴ where the prognosis was grave (mortality 40%) which could be because of variation in plasmodial species and different genetic constitution and susceptibility of the population in two different continents.

Autonomic involvement may be a cause of sudden death in patients of falciparum malaria besides seizures, hypoglycaemia, dyselectrolytaemia, quinine/drug induced arrhythmia, multiorgan dysfunction, etc.

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