Severe myoclonus in a patient recovering from falciparum malaria

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Isolated myoclonus has rarely been reported as a complication of Plasmodium falciparum *malaria. We describe the development of chaotic myoclonic jerks in an afebrile and conscious patient, the fourth day of treatment with quinine for* P. falciparum *infection. The myoclonus finally resulted in a generalized tonic-clonic seizure and coma, which resolved without further antimalarial treatment.*

Key words: myoclonus, malaria

In adults cerebral malaria generally presents as an acute diffuse encephalopathy in the 2nd and 3th week of the *Plasmodium falciparum* infection with abnormal behaviour, diminished consciousness, coma or convulsions which mostly are generalized. Myoclonus has rarely been described as a complication of malaria.[1,2]

CASE REPORT

A 29-year-old Belgian businessman was admitted to our hospital. During the last 8 years he had frequently visited Sierra Leone but never took any malaria prophylaxis. Seventeen days after his last stay he developed chills, a dry mouth, dyspepsia and general weakness. A thick smear showed presence of *P. falciparum*. Quinine sulphate and doxycycline were started. Four days later he was admitted to the hospital with violent irregular asynchronous myoclonias in both arms and legs. He was well-oriented and alert. The temperature was 37°C, the pulse 92/min and the blood pressure 100/70 mmHg. Neck stiffness, trismus and photophobia were absent. No malaria parasites were present in a thick smear. Serum glucose level was 137 mg/dl, natrium 136 mmol, potassium 5.1 mmol/l and calcium 9.7 mg/dl. Antimalaria treatment was stopped. Five hours after the onset of the myoclonus he developed a generalized tonic-clonic seizure with urine-loss but without tongue bite, controlled with diazepam 20 mg IV. A CT scan of the brain did not reveal structural abnormalities. Analysis of the cerebrospinal fluid showed a normal cell count and glucose level but an elevated protein concentration (118 mg/dl). After the seizure the patient remained comatous (Glasgow coma scale, GCS, E1V1M1). Intravenous diphantoine, amikacin, ticarcillin and acyclovir treatment were started. Ten hours after the onset of the coma he reacted to speech but was unable to follow instructions (GCS E3V1M3). Twenty hours after admission he regained consciousness and the clinical examination was normal. Five days after admission a thick smear was again positive for *P. falciparum*. Halofantrine 1500 mg/12 h was given orally and repeated one week later. Seizures did not recur nor did the myoclonus, despite withdrawal of diphantoine.

DISCUSSION

Appearance of myoclonias late in the course of malaria infection has been described by Daroff, but altered consciousness and fever were present.[3] In our patient, the myoclonias were almost certainly due to malaria: the patient never had seizures before or after this illness, his family was free of neurologic disease, the patient reported no drugs or other medication intake, the diagnosis of *P. falciparum* malaria was proven twice, a partially treated meningitis (with doxycycline, given as antimalarial treatment) is unlikely in view of the normal cellular count of the CSF on two occasions, and viral encephalitis is frequently associated with typical CT scan images and would never respond so dramatically to antiviral therapy. Serum levels of quinine were not determined, but myoclonias have not been reported as an adverse effect of quinine[4], and quinine is commonly prescribed for muscular cramps.

In conclusion, this case report suggests that violent extensive myoclonias may occur late in the course of an already treated *P. falciparum* malaria infection, even in the presence of a negative thick smear, and that they can herald cerebral malaria.

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