Lesson of the month 1: Post-malaria neurological syndromes

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There are three neurological syndromes that may follow malarial infection after recovery and at a time when the patient is aparasitaemic. An acute disseminated encephalopathy, a cerebellar syndrome, and an acute demyelinating polyneuropathy. This paper reports a 42-year-old male patient who developed encephalopathy.

**Key words**: Plasmodium falciparum malaria, post-malaria syndromes, encephalopathy

**Introduction**

There are three neurological syndromes that may occur after recovery from malaria, with no parasitaemia, usually following Plasmodium falciparum malaria and occurring after an interval of 2 days to 2 months. The syndromes are an acute disseminated encephalopathy, known as post-malaria neurological syndrome (PMNS); a delayed cerebellar syndrome; and an acute idiopathic demyelinating polyneuropathy (AIDP). These syndromes may follow recovery from an attack of *falciparum* malaria with no parasitaemia, and more commonly occurs after treatment with mefloquine.

**Case history**

A 42-year-old right-handed commercial airline pilot, who spent one night in West Africa, had a mosquito bite on his left arm while having a drink in the evening at the hotel poolside. He had applied a topical anti-mosquito repellent containing diethyltoluamide (DEET), but he had not taken any oral anti-malarial prophylactic medication. Five days later he became unwell with flu-like symptoms and headache. These symptoms progressed over the next 3 days, and following his return home he attended his GP. Malaria was suspected and he was admitted to hospital where *P falciparum* malaria was confirmed with a parasitic load of 5.6%. He spent 5 days in the intensive care unit, treated with intravenous quinine for 2 days then oral quinine, oral doxycycline and an exchange transfusion. Complications included renal impairment with a raised urea and creatinine, anaemia, thrombocytopenia and meralgia paraesthetica. He was discharged after 8 days on ferrous fumarate and omeprazole. When seen as an outpatient 9 days later, he was found to be anaemic (haemoglobin 10.7 g/dL).

**Discussion**

The World Health Organization estimates that in 2013 there were 214 million cases of malaria worldwide, with 438,000 deaths. Malaria is endemic in 97 countries with 3.2 billion people at risk, and 1.2 billion at high risk. It is encouraging that there has been a 47% reduction in mortality since the start of this century. Considering this very large number of cases across the world, it is very surprising that PMNS was
only first described in 1996, and since then there have been fewer than 50 cases reported in the literature, mostly as single case reports. It therefore appears to be an extremely rare complication but it may also be grossly underreported, since a large number of those affected are young children in the developing world and once recovered from malaria, patients may return home and the subsequent illness either not reported or not ascribed to a complication of malaria.

There appear to be three recognised PMNSs, which are defined as a neurological syndrome that develops after recovery from malaria at a time when the patient is aparasitaemic. This nearly always follows *P falciparum* infection. The first syndrome to be described was delayed cerebellar ataxia in 1986, followed by Guillain–Barre–Strohl Syndrome (AIDP) in 1992, and lastly PMNS in 1996.

The latter report remains the largest and most definitive series with 19 adults and three children identified out of 18,124 patients with *falciparum* malaria; of whom 1,176 (6.5%) were severely affected. The clinical features of PMNS as defined in this and subsequent reports are: onset at 2–60 days after recovery from *P falciparum* malaria; mean duration of the encephalopathy was 60 hours (24–240); self-limiting without treatment, though steroids may help. In the acute phase there may be rapid onset with 19 adults and three children identified out of 18,124 patients.

References

5. AIDP

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