Plasmodium vivax Cerebral Malaria

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We report two cases of Plasmodium vivax malaria (both aged 12 years) complicated by seizures and symptoms of diffuse meningoencephalitis. One had predominantly meningeal signs while in the other, purely encephalitis features were present. Both cases were treated with artesunate. Rarely, cerebral malaria is a presenting complication or occurs during the course of P. vivax infection.

Key words: *Cerebral malaria, Meningoencephalitis, Plasmodium vivax, Seizures.*

Cerebral malaria is usually secondary to *P. falciparum* infection. However, there are infrequent reports of cerebral malaria associated with *P. vivax* infection. To our knowledge, only 45 cases of central nervous system *P. vivax* malaria are reported in the English literature since 1920; about half of these cases have occurred in children(1,2).

Case Report

Two boys, each aged 12 years presented with high grade intermittent fever of more than 4 days duration. Both of them were in altered consciousness at the time of admission. There was history of generalized tonic clonic convulsion prior to admission in each case. Both were severely dehydrated. Their capillary blood sugar level was normal at presentation. Beside these, the differentiating features between the two are depicted in *Table 1*.

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In both the cases, routine count, liver function tests and serum electrolytes were within normal limits. Peripheral blood smear revealed trophozoites of *P. vivax*. Antigen test (OptiMAL) for *P. vivax* was positive while that for *P. falciparum* was negative in both the cases. Their cerebro-spinal fluid and electroencephalogram (EEG) findings were normal. They were put on supportive therapy and intravenous artesunate in the recommended dose. Repeat blood smear after 2 days showed clearance of the parasite. Both were discharged in a clinically stable condition and advised primaquine for 14 days. Follow up after one month showed no residual neurological deficit.

Discussion

Organ dysfunction characteristic of *P. falciparum* malaria is unusual in *P. vivax* infections. Any patient infected with *P. vivax* who exhibits severe malaria is presumed to be suffering from mixed infection(2). However, that may not be always true. As evident from the present report, *P. vivax* infection can also present as cerebral malaria.

Clinical data provided by Kochar, *et al.* indicates that *P. vivax* can cause both sequestration-related and non-sequestration related complications of severe malaria, all of which are commonly associated with *P. falciparum* infections(3). The exact pathogenetic mechanism however remains elusive. Sachdev and Mohan(4) studied the clinico-laboratory profile of six patients with vivax cerebral malaria. The presenting features were of an acute febrile encephalopathy, convulsions and coma. Focal neurological signs were observed in one patient. Ozen, *et al.*(1) have recently described a case of cerebral vivax malaria that presented with status epilepticus.

Some experts also suggest that cerebral malaria subjects might have an underlying seizure disorder and those seizures are precipitated by the high fever associated with the disease.

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TABLE I–*Differentiating Features Between the Two Cases*

	Case 1	Case 2
Presenting features:	1. Deteriorating consciousness for 24 hr 2. Generalized convulsion-1 episode 3. Retention of urine 4. Recovery – 10 hr	1. Unconsciousness at the time of admission 2. Generalized convulsion-3 episodes 3. Recovery – 15 hrs
Examination at presentation:	EMV score – 11 Meningeal signs present Plantars – extensor bilaterally Pupils – normal Liver – 1 cm palpable Spleen – 6 cm palpable	EMV score – 5 Meningeal signs absent Plantars – extensor bilaterally Pupils – mid dilated No hepatosplenomegaly
Investigations:	Hemoglobin – 6 g% Renal parameters: normal	Hemoglobin – 13.7 g% Urea and creatinine: increased

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