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LETTERS TO EDITOR

ACUTE DISSEMINATED ENCEPHALOMYELITIS AFTER TREATMENT OF SEVERE FALCIPARUM MALARIA

Sir,

A 20-year-old female showed a comatose state for 5 days. Previously she had been treated for falciparum malaria with parenteral quinine 1 gm loading dose followed by 500 mg thrice a day for 8 days. On the sixth day, she developed altered consciousness. On arrival, the temperature was 38°C, pulse 126/min, respiratory rate 24/min and blood pressure 120/70 mmHg. Chest, cardiovascular system and the abdomen were normal. Neurological examination disclosed coma, decerebration, hypertonia, hyperreflexia and Babinski's responses. Laboratory tests showed anemia (6.7 gm%), and the serum biochemistry was normal. The peripheral blood smear revealed dual-deficiency anemia, and no malarial parasites were seen. Cerebrospinal fluid examination, 100 lymphocytes/µl; proteins 45 mg/dl; and glucose level 84 mg/dl. MRI of the brain showed diffuse white matter hyper-intensities on T2 images, involving the subcortical white matter, corpus callosum and midbrain. Treatment with empirical broad-spectrum antibiotic was started (injection Cefepime 2 g BD with Teicoplanin 300 mg BD), and 2 units of blood were transfused. She remained deeply comatose and febrile; and after the MRI scan, intravenous methyl-prednisolone 1 g per day was administered for 3 days. The fever subsided and the patient showed a marked neurological improvement.

Postmalarial neurological syndrome is defined by an acute onset of neurological abnormalities in patients recently recovered from severe *Plasmodium falciparum* malaria. The lack of malarial parasite on peripheral blood smear examination distinguishes postmalarial neurological syndrome from cerebral malaria. The time from eradication of the parasitemia to development of this syndrome varies from 4 days to 9 weeks.[1] Clinical features are convulsions, confusion-like state, psychosis, cerebellar ataxia, aphasia and myoclonus.[1]

Acute disseminated encephalomyelitis is an acute demyelinating disease occurring 1-3 weeks after a viral infection or vaccination.[1,2] The illness of our patient began after severe falciparum malaria complicated by a rapidly oncoming demyelinating illness after anti-malarial therapy. To the best of our knowledge, there are only three cases of ADEM following treatment of malaria. The first had neurologic dysfunction, seizures and hyper-intense lesions in subcortical white matter on T2-weighted MRI images, after recovering from falciparum malaria.[2] The second case had neurological disturbances and multiple high-intensity lesions in the brain after recovery from *Plasmodium vivax* infection.[3] The third case presented with an ADEM-like illness after treatment of falciparum malaria. Identification of Varicella-Zoster virus infection using polymerase chain reaction on cerebrospinal spinal fluid suggested infection.[4] The neurological syndrome in our patient occurred after anti-malarial therapy and

responded to corticosteroids. MRI abnormalities reported in cerebral malaria include cortical infarcts or focal or diffuse white matter hyper-intensities. In ADEM, MRI shows diffuse T2 hyper-intensities involving the subcortical white matter, corpus callosum and brain stem.[5] MRI findings in our patient were suggestive of ADEM.

ADEM can be regarded as a neurological complication of severe falciparum malaria. Raised levels of neurotoxic cytokines in the cerebrospinal fluid, tumor necrosis factor-α and interleukins 2 and 6 have been seen in patients with severe malaria and PMNS.[6] The latency to neurological involvement after eradication of the parasite and the response to steroid treatment in our patient support an immunological mechanism.

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