EDITORIAL

Cerebral Malaria

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S. K. Gupta, Annil Mahajan

Malaria is one of the oldest disease known to the mankind as it is evident from the scriptures of Egypt, Indonesia and China. Hippocrates (460-375 BC) described the symptoms as periodic fever-quotidian, tertian, quartan and sub-tertian.

Malaria is a protozoal disease characterized by fever, splenomegaly, anaemia and chronic relapsing course. Since 1956 when national Malaria Eradication Programme (NMEP) was sponsored by WHO, there were impressive results initially in the containment of the disease. However, due to various factors like resistance to insecticides and anti-malarial drugs, failure of supressive therapy, there has been resurgence of the disease in many parts of the world. Consequently, malaria remains today, as it has been for centuries, one of the most serious disease in the world especially in the tropical countries. Malaria continues to be a major global health problem with over 40% of the world population and more than 2000 million people exposed to varying degrees of malaria risk in 100 countries (1). In addition with modern rapid means of travel, large number of people from non-malarial areas are being exposed to infection.

India is a vast subcontinent having varied climate and topography. The Indo-Gangnetic plains have variable rainfall and are prone to epidemics. Because of resistance to insecticides as well as drug resistant strains especially of plasmodium falciparum throughout South Asia, Western Pacific, Central and South America, falciparum malaria is encountered in these regions. Sudies carried out in India (2-5) revealed falciparum malaria resistent to chloroquine to be widespread in North Eastern and Eastern regions (Assam, Arunachal Pradesh, Manipur, Meghalaya, Mizoram, Nagaland, Tripura, Orissa and West Bengal). One hundred and ten new foci of drug resistance were added in 1987 (6). Thar region in Rajasthan is now experiencing the epidemics of cerebral malaria. As a result of the increase in drug resistance in our country the incidence of complicated forms of malaria is increasing and hence cerebral malaria poses formidable challenge.

Central nervous system (CNS) involvement in malaria usually occurs with P. falciparum infection. CNS involment is reported to occur in 2-55% cases in different series (7,8). Cerebral malaria is described as a serious acute febrile encephalopathy with no localizing signs or transent neurological signs as a rule. However, clinical presentations of cerebral malaria are protean and can be as cerebral or spinal syndromes, peripheral neuropathy and acute psychiatric manifestations. Late sequlae include epileptic disorders, extrapyramidal syndromes (dyskinesias, chorea parkinsonism) besides psychiatric disorders.

The cerebral syndrome is characterized by acute febrile encephalopathy including coma, cranial nerve palsies, hemiplegia, paraplegia and extrapyramidal involvement. The spinal disorders include the picture of amyotrophic lateral sclerosis, funicular-myelosis, spastic spinal paralysis and dorsalis tabes. In peripheral nervous system the manifestations are neuritis, polyneuritis and G. Barre syndrome like picture have been seen (9).

Cerebral malaria should be suspected in a patient living in the tropics, especially in endemic areas, presenting with acute encephalopathy, neuro-psychiatric

From the Postgraduate Department of General Medicine, Government Medical College, Jammu (J&K), India.

Correspondence to : Dr. S. K. Gupta, Associate Prof. & Consultant Neurologist, Government Medical College, Jammu (J&K), India.

manifestations or presenting with a history of blood transfusion. Diagnosis is established by demonstration of parasite in peripheral blood reported at 4-6 hours and at peaks of fever (over 104°F) or infrequently by sternal puncture. The presence of malarial pigment in monocytes is a useful indicator of diagnosis of malaria aespecially in anaemic children and in patients with severe malaria associated with absent or low parasitaemia. C.S.F. findings may be variable and are not characteristic in cerebral malaria.

Cerebral malaria has to be differentiated from other causes of coma specially metabolic–diabetic, uraemic, hepatic or alcoholic. Infections like acute meningitis, encephalitis, brain abscess, miliary tuburculosis or any other cause of PUO should be excluded by C.S.F. examination and other appropriate investigations including imaging procedures.

Management should be aimed at reduction of parasitaemia by quinine hydrochloride or chloroquine. In chloroquine resistant malaria quinine is the drug of choice. The dose of quinine needs to be slightly reduced if there is associated renal failure since a small proportion (20%) is eliminated in the urine (10).

Recently, continuous infusion of quinidine gluconate (11) has been found to be more effective in treatment of severe malaria including cerebral malaria. Care should be taken for recording Q.T. interval. Other drugs used for cerebral malaria are artemether and artesunate. Supportive therapy should be provided to correct fluid and electrolyte balance, control of convulsions, cerebral oedema and hyperpyrexia. Complications of severe malaria like black water fever, disseminated intravascular corgulation, acute renal failure and hepatic failure will also need attention and appropriate therapy.

In conclusion cerebral malaria has protean manifestations and any axis of the CNS may be implicated (14). Early and prompt treatment of th disease and its complications is rewarding.

References

1. Management of Severe and complicated Malaria - A Practica Hand Book (ed) Gilles HM. WHO Geneva 1991.
2. Sehgal PN, Sharma MID, Sharma SL, Gogai S. Resistanc of chloroquine in falciparum malaria in Assam State. *Ind J Comm Dis* 1973 ; 5 : 175-80.
3. Kunte AB, Mitra NK. Prevalence of chloroquine resistanc in malaria. *J Assoc Phys Ind* 1978 ; 26 : 41-48.
4. Gopinathan VP, Dutta PK, Bhupte AG. Falciparum malari in North Eastern states. *J Assoc Phys Ind* 1981 ; 29 : 1029.
5. Dhamija RM, Venkataraman S. Diagnosis and management of cerebral malaria. *Neurosciences Today* 2003 ; 7 : 70-78.
6. The clinical management of acute malaria. WHO Regional publication. *South East Asia Series* 1988 ; 8.
7. Dhamija RM, Vankataraman S, Banerjee AK. Cerebral malaria. In : Aduanes in clinical medicine-I, (ed) Ahuja MMS. BT Churchill Livingstone, New Delhi 1991 ; 1-27.
8. Mehta SR, Naidu G, Chander V, Singh IP, Johri S, Ahuja RC. Falciparum malaria present day problem. An experience with 425 cases. *J Assoc Phys Ind* 1989 ; 37 : 264.
9. Arya TVS, Prasad RN. Falciparum malaria presenting as Guillian Bare syndrome. *Br Med J* 1986 ; 292 : 1430.
10. Davis TME, Supranaranond W, Pukrittayakamee S. A safe and effective consecutive infusion regimen for rapid quinine loading in severe falciparum malaria. *J Infect Dis* 1990 ; 161 : 1305-08.
11. Miller KD, Greenberg AE, Campbell CC. Treatment of severre malaria in United States with a continuous infusion of quinidine gluconate and exchange transfusion. *N Engl J Med* 1989 ; 321 : 65.
12. Hein TT, Day NPJ, Phu NH. A controlled trial of artemether or quinine in Vietnamese adults with severre falciparum malaria. *N. Engl J Med* 1996 ; 335 : 76-83.
13. De Vries, Dien TK. Clinical pharmacology and therapeutic potential of artemisinin and its derivatives in the treatment of malaria. *Drug* 1996 ; 52 : 818-36.
14. Garg RK, Karak B, Misra S. Neurological manifestations of malaria. An update. *Neurology India* 1999 ; 47 85-91.