Cerebral Malaria

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Malaria is one of the oldest disease known to 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 suppressive 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-Gangetic 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. Studies carried out in India (2-5) revealed falciparum malaria resistant 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). That 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 involvement 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 transient 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 sequelae 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, fumricular-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

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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 especially 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 tuberculosis 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 coagulation, 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 the disease and its complications is rewarding.

References