BMC Proceedings



Oral presentation

Open Access

Plasmodium falciparum and the brain Charles RIC Newton

Address: Kenya Medical Research Institute/Wellcome Trust Collaborative Programme, P.O. Box 230, Kilifi, 80108 Kenya and Institute of Child Health, University College London, UK

Email: Charles RJC Newton - cnewton@kilifi.kemri-wellcome.org

from Infectious diseases of the nervous system: pathogenesis and worldwide impact Paris, France. 10-13 September 2008

Published: 23 September 2008

BMC Proceedings 2008, 2(Suppl 1):S34

This abstract is available from: http://www.biomedcentral.com/1753-6561/2/S1/S34

© 2008 Newton; licensee BioMed Central Ltd.

Over 2 billion people are exposed to *Plasmodium falciparum* infections, with 500 million infections per year. Children living in malaria endemic areas of sub-Saharan Africa bear the brunt of the disease. In malaria endemic areas, severe falciparum malaria usually develops after 6 months of age.

A unique characteristic of *P. falciparum* is that the infected erythrocytes sequester within the deep vascular beds, particularly those of the brain. The blood brain barrier appears to be impaired. The most common CNS manifestations are seizures, agitation, psychosis, impaired consciousness and coma (cerebral malaria), but there are differences in clinical presentation between African children and non-immune adults.

During the acute infection, *P. falciparum* appears to be epileptogenic: convulsions are more common falciparum than vivax malaria despite a similar febrile response; over half convulsions occur when the child is afebrile; 86% have complex features, much higher than any reports of febrile seizures. Falciparum malaria is the most common cause of status epilepticus in these areas.

Cerebral malaria is the most severe neurological complication associated with a mortality of 13–17%. Seizures occur in over 80% of children with cerebral malaria, and prolonged seizures are associated with neurocognitive sequelae. The mechanisms of impaired consciousness are unknown, although microvascular obstruction and neurotoxins such as cytokines and quinolinic acid are thought to contribute.

About 11% in children with cerebral malaria discharged from hospital with neurological deficits. Subsequently many of these deficits improve, but 10% of children develop epilepsy 3–6 years following cerebral malaria and 24% of children have neurocognitive deficits when assessed during school age. The cause of the neurological damage is unknown, but it is associated with seizures and intracranial hypertension, and there is evidence of axonal and astrocyte damage. Erythropoietin appears to protect the brain against neurological damage.

Given the magnitude of *P. falciparum* infection in the world, falciparum malaria may the most common infection of the CNS, and a major cause of disability and epilepsy. Further research to elucidate the mechanisms of neurological involvement and damage are required.