BMC Proceedings

BioMed Central

Poster presentation

Open Access

Cognitive deficits following exposure to malaria with neurological involvement: an event related potentials study

Michael Kihara*[1], Michelle de Haan[2], Harrun H Garrashi[1], Brian G Neville[3] and Charles R Newton[1,3,4]

Address: [1]The Centre for Geographical Medicine Research (Coast), Kenya Medical Research Institute, Kilifi, Kenya, [2]Developmental Cognitive Neuroscience Unit, University College London, Institute of Child Health, UK, [3]Neurosciences Unit, University College London Institute of Child Health, The Wolfson Centre, UK and [4]London School of Hygiene and Tropical Medicine, London, UK

Email: Michael Kihara* - mkihara@kilifi.kemri-wellcome.org

* Corresponding author

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):P30

This abstract is available from: http://www.biomedcentral.com/1753-6561/2/S1/P30

© 2008 Kihara et al; licensee BioMed Central Ltd.

Introduction

Plasmodium falciparum is the most common parasitic infection of the nervous system. It affects about 500 million people worldwide and causes up to 1 million deaths each year mostly children under 5 years old in Africa. It causes neurological complications which are associated with persisting cognitive deficits in between 5–21% cases. However, a lack of sensitive, culture-fair and robust techniques of data collection in the rural settings prevented accurate estimate of the neuro-cognitive burden of severe malarial disease. We proposed the use of auditory and visual event related potentials (ERPs) as a marker of cognition.

Methods

Fifty-four children, aged between 6–7 years, who had been hospitalized at Kilifi District Hospital (KDH) with severe forms of falciparum malaria were selected and compared with an equal number of unexposed children selected from the community. We recorded passive auditory and visual event related potentials in each of these children.

Results

The results showed that children exposed severe falciparum malaria had significantly longer auditory N200 latencies (252 ± 48 vs 183 ± 42, $p < 0.01$) and P3a latency (330 ± 55 vs 305 ± 52, $p < 0.05$) compared to community controls. Children with a history of severe falciparum malaria had smaller P3a amplitudes (0.2 ± 5.9 vs 6.2 ± 7.7, $p < 0.05$) compared to controls. In the visual paradigm, children with a history of malaria plus seizures had significantly smaller P200 amplitudes (3.2 ± 15.8 vs 5.5 ± 7.6, $p < 0.05$) than community controls.

Conclusion

The results from the ERP tests demonstrate that children with a history of severe falciparum malaria could have slower processing than community controls. The pattern of deficits in cerebral malaria is different from those with malaria plus seizures and prostrated malaria. The results provide further further evidence of deficits in areas of attention, stimulus classification and discrimination and most likely localized to the superior temporal gyrus or prefrontal cortices.