CEREBRAL MALARIA IN CHILDREN

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INTRODUCTION

- A major public health problem in the developing world

- Over 70% of new infections worldwide occur in children living in SSA.

- Children are susceptible to developing cerebral malaria.
Cerebral malaria is:

- The most severe neurological complication
- Mortality of 15–20%
- Substantial proportion of individuals with this condition develop neurocognitive sequelae
PATHOGENESIS

- P. falciparum the main culprit

- Cytoadherence and sequestration of erythrocytes in microvasculature

- Mediated by proteins encoded by the var genes of the parasite

- Proteins act as ligands to proteins in the venules
cytoadherence

PfEMP-1 (var genes) as Parasite Ligand

Antigenic Variation

Endothelial Cell Receptors
sequestration is due to;

- Autoagglutination –btn infected rbcs
- Rosseting –btn uninfected rbcs
- platelet-mediated clumping
Platelet mediated clumping
Sequestration reduces microvascular flow with resultant ischaemic injury.

Occurs in the context of severe anemia, hypoglycemia and increased basal metabolism due to fever.
- Cytokine theory – both pro(tnf & il-6) and anti(il-10) inflammatory.

- Induce inducible NO synthase in ec in the brain – increased NO causes exitotoxicity.
- All children with cerebral malaria have increased ICP.

- Caused by brain edema

- Potentially lethal with neurological sequelae.
Diagnosis

- any patient who has a febrile illness with neurological symptoms and has passed through a malaria endemic area in the past months

- Microscopy or immunodiagnosis

- In malaria-endemic areas, falciparum malaria is a diagnosis of exclusion
Neurological sequelae

- between % and % at the time of hospital discharge
- May be transient or persist for life
- Include cortical blindness, hemiparesis, cerebellar ataxia, psychosis and epilepsy
Subtle deficits (for example, cognitive difficulties, language and behavior problems) are increasingly recognized, and have been documented in % of children after recovery from cerebral malaria.
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