ABSTRACT

Cytoadherence of *Plasmodium falciparum*-infected erythrocytes to the brain microvascular endothelial cells is believed to be important cause of circulatory blockage in cerebral malaria. Cytokines released during the acute infection can activate endothelial cells leading to increase binding of infected erythrocytes due to subsequent increase of adhesion proteins. This effect may be direct and more potent with the tissue-localized cytokines in the brain. In this study paraffin-embedded brain tissues of cerebral, and of noncerebral malaria were compared. The difference in histopathologic changes of each case was clearly shown. The results demonstrated that tissue-localized TNF-α, IFN-γ, IL-1β, and IL-10 in the brain were associated with cerebral malaria. IL-4 was the only cytokine presented at high level in the brain tissue of noncerebral malaria. No elevation of the adhesion proteins could be demonstrated in the brain tissues of all malaria cases.