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The levels of CD16/Fc gamma receptor III A on $CD14^+CD16^+$ monocytes are higher in children with severe *Plasmodium falciparum* anemia than in children with cerebral or uncomplicated malaria

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Background

Fc gamma receptor IIIA (CD16/FCγRIIIA) on monocytes/macro-phages may play an important role in the pathogenesis of severe malarial anemia (SMA) by promoting phagocytosis of IgG-coated uninfected red cells and by allowing the production of tumor necrosis factor alpha (TNF-α) upon cross-linking by immune complexes (ICs). However, not much is known about the differential expression of this receptor on monocytes of children with severe malaria and uncomplicated malaria.

Materials and methods

We investigated the expression of CD16/FCγRIIIA on monocytes of children with SMA, cerebral malaria (CM), and their age-matched uncomplicated malaria controls by flow cytometry. Since CD14low (CD14+) monocytes are considered more mature and macrophage-like than CD14high (CD14++) monocytes, we also compared the level of expression of CD16/FCγRIIIA according to the CD14 level and studied the relationship between CD16/FCγRIIIA expression and intracellular TNF- production upon stimulation by ICs.

Results

CD16/FcγRIIIA expression was the highest overall on $CD14^+CD16^+$ monocytes of children with SMA at enrollment. At convalescence, SMA children were the only ones to show a significant decline in the same parameter. In contrast, there were no significant differences among groups in the expression of CD16/FCγRIIIA on $CD14^{++}CD16^+$ monocytes. A greater percentage of $CD14^+CD16^+$ monocytes produced TNF-α upon stimulation than any other monocyte subset, and the amount of intracellular TNF-α correlated positively with CD16/FcRIIIA expression. Furthermore, there was an inverse correlation between hemoglobin levels and CD16/FCγRIIIA expression in children with SMA and their controls.

Conclusions

These data suggest that monocytes of children with SMA respond differently to *Plasmodium falciparum* infection by overexpressing CD16/FCγRIIIA as they mature, which could enhance erythrophagocytosis and TNF production.

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