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Plasmodium falciparum and placental cytokine profiles among pregnant women in relation to their HIV-1 status: possible implications for mother-to-child transmission (MTCT) of HIV-1 in Cameroon

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Background

Placental cytokines play vital roles in establishing and maintaining pregnancy as well as protecting the fetus from *in utero* infections. Previous studies have strongly suggested the implication of co-infections such as *P. falciparum* in the *in utero* MTCT of HIV-1 [1][2][3]. This study was designed to assess the impact of *P. falciparum* on the influence of HIV-1 infection on placental cytokine profile and the association of these profiles with clinical factors known to be related to HIV-1 MTCT.

Materials and methods

P. falciparum was tested in the peripheral and/or placental blood from 50 and 80 HIV-1 negative and positive women respectively. Cytokines (proteins) were quantified in the supernatants of 24 hours culture of placental explants by ELISA while cytokine mRNAs were quantified in placental tissue by real time PCR. Antibodies to the DBL3γ domain of PfEMP1 that binds *P. falciparum* infected red blood cells to placental CSA were titrated by ELISA in sera. The comparisons of the levels of cytokine proteins and mRNAs, as well as of anti-DBL3γ antibodies between HIV-1 negative and positive women who were either *P. falciparum* negative, or positive in the periphery or placenta, were tested through non-parametric tests, as well as the associations between cytokine profiles and clinical factors.

Results

Placental *and peripheral P. falciparum* infection was comparable in both HIV-1 negative and positive women (from 18 to 24%). Conversely *P. falciparum* parasitemia was significantly higher in the HIV-1 positive group. Large individual variations were observed in placental cytokine proteins and mRNA expression in each group. No significant differences were observed between placental cytokine median levels (protein and mRNA) in HIV-1 negative and positive women. However, among *P. falciparum* negative women, we observed significant differences in several cytokine median levels (TNF-α, IL-10, IL-16, IL-7, LIF, and RANTES) between HIV-1 negative and positive women. Median levels of antibodies to DBL3γ were significantly higher in the HIV-1 negative group (p=0.03) and was dependent of peripheral and placental *P.falciparum* infection. TNF-α among the HIV-1 positive women was the only cytokine associated with clinical parameters linked to HIV-1 MTCT (premature rupture of membranes, number of pregnancies and parity; p≤0.04).

Conclusions

Altogether these results highlight the reciprocal influence of both infections at the materno-fetal interface that might have possible implications for *in utero* HIV-1 MTCT in areas where HIV-1 and *P. falciparum* co-circulate.

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