P1261
Poster Session V
Immunology, vaccination and host defences

ASSOCIATION OF MATERNAL HIV AND MALARIA WITH REDUCED PLACENTAL TRANSFER OF ANTIMALARIAL ANTIBODIES

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INTRODUCTION: During pregnancy, maternal antibodies are transferred to the fetus by an active process in the placental syncytiotrophoblast that contributes to minimize deficiencies in antibody production during the first months of infants' life. Previous studies suggest that malaria and human immunodeficiency virus (HIV) infections during pregnancy affect this placental transfer, although their effect over specific antimalarial antibodies, as well as the effect of undiagnosed submicroscopic malaria infections, remains unclear.

OBJECTIVES: Therefore, this study was aimed to investigate maternal factors affecting the transplacental transfer of a wide range of antimalarial antibodies from mother to fetus and its potential relationships with adverse outcomes at delivery and malaria incidence in the newborn during the first year of life.

METHODS: Total immunoglobulin (IgG) and IgG subtype levels against *Plasmodium falciparum* antigens merozoite surface protein-1-19 ($MSP1_{19}$), erythrocyte binding antigen 175 (EBA175) and apical membrane antigen 1 (AMA1) and against parasite lysate were measured by ELISA in 187 mother-cord pairs from Mozambique. Placental antibody transfer was defined as the Cord-to-Mother Ratio of the antibody levels. Malaria diagnosis relied on thin and thick smears for peripheral maternal and cord blood. Placental malaria was defined by histological examination. Detection of *P. falciparum* submicroscopic infections was performed by quantitative PCR in all the samples and maternal HIV status was determined by using two different rapid diagnostic tests.

RESULTS AND DISCUSSION: Maternal HIV, peripheral and placental malaria, including submicroscopic infections, were associated with a significant decrease in the levels of antimalarial antibodies in cord blood and with reduced placental transfer of antimalarial antibodies from mother to fetus. In a multivariate analysis, maternal HIV was associated with reduced CMR of IgG1, IgG2 and IgG3 against lysate; IgG1 and IgG4 against AMA1; IgG3 and IgG4 against $MSP1_{19}$; and IgG3 against EBA175. In the periphery, both microscopic and submicroscopic malaria infections were associated with reduced CMR of IgG1 against lysate; and submicroscopic infections with reduced CMR of IgG3 against $MSP1_{19}$ and IgG4 against EBA175. In the placenta, submicroscopic malaria infection was associated with reduced CMR of IgG, IgG3 and IgG4 against EBA175. In general, higher transfer of antimalarial antibodies was not associated with adverse outcomes in the infant or reduced risk of malaria during the first year of life, pointing towards these antibodies as markers of exposure during pregnancy rather than protection.

CONCLUSIONS: Maternal HIV and malaria, including submicroscopic infections, reduce the placental transfer of antimalarial antibodies from mother to fetus. Further studies are needed to evaluate the association between antibody transfer and malaria risk in infants that helps to the design of effective control strategies and adequate diagnostic and treatment tools for malaria and HIV in pregnancy.