Abstract

The role of *Plasmodium falciparum* malaria in Epstein–Barr virus (EBV) transmission among infants early in life remain elusive. We hypothesized that infection with malaria during pregnancy could cause EBV reactivation leading to high EBV load in circulation, which could subsequently enhance early age of EBV infection. Pregnant women in Kisumu, where *P. falciparum* malaria is holoendemic, were actively followed monthly through antenatal visits (up to 4 per mother) and delivery. Using real-time quantitative (Q)-PCR, we quantified and compared EBV and *P. falciparum* DNA levels in the blood of pregnant women with and without *P. falciparum* malaria. Pregnant women that had malaria detected during pregnancy were more likely to have detectable EBV DNA than pregnant women who had no evidence of malaria infection during pregnancy (64 vs. 36 %, $p = 0.01$). EBV load as analyzed by quantifying area under the longitudinal observation curve (AUC) was significantly higher in pregnant women with *P. falciparum* malaria than in women without evidence of malaria infection ($p = 0.01$) regardless of gestational age of pregnancy. Increase in malaria load correlated with increase in EBV load ($p < 0.0001$). EBV load was higher in third trimester ($p = 0.04$) than first and second trimester of pregnancy independent of known infections. Significantly higher frequency and elevated EBV loads were found in pregnant women with malaria than in women without evidence of *P. falciparum* infection during pregnancy. The loss of control of EBV latency following *P. falciparum* infection during pregnancy and subsequent increase in EBV load in circulation could contribute to enhanced shedding of EBV in maternal saliva and breast milk postpartum, but further studies are needed.