ABSTRACT
Malaria and human immunodeficiency virus (HIV) are co-endemic in sub-Saharan Africa. Infection with HIV results in B cell anomalies. Antibodies are critical in protection against malaria and it is hypothesized that B cell anomalies resulting from HIV infection interfere with antibody responses contributing to severe and frequent malaria episodes. Individuals infected with HIV have elevated antibody and C-reactive protein (CRP) levels. However, it is unclear whether malaria-specific antibodies, particularly immunoglobulin M (IgM), total immunoglobulin G (IgG) and IgG subclasses levels would be increased or decreased, given the evidence of impaired B cell responses to other antigens due to HIV. Furthermore, how malaria-specific antibodies correlate with viral load (VL) and CRP levels in HIV infected individuals is unclear given their link in HIV disease progression. *Plasmodium falciparum* (*Pf*) vaccines studies have associated malaria protection with antibodies against apical membrane antigen-1 (AMA-1) and glutamate-rich protein-R0 (GLURP-R0). The current study aimed to: determine quantities and prevalence of antibody isotypes (IgG and IgM) against *Pf* antigens (AMA-1 and GLURP-R0); determine quantities and prevalence of IgG subclasses in response to *Pf* selected antigens and; measure the correlation of *Pf* specific antibody isotypes and subclasses with VL, CD4+ counts and CRP levels. A comparative cross-sectional study using a sample size of 181 comprising of 52 HIV negative and 129 HIV positive adult participants seeking care at Bondo sub-County Hospital was conducted. Data from Bondo Sub-County hospital have shown an overlap in malaria and HIV infections. Antibody and CRP levels were tested using ELISA. The CD4+ cell and VL counts were obtained using FACSCount and Abbott m2000 analyzer respectively. Medians and proportions of *Pf*-specific antibody levels were compared using Wilcoxon Rank-Sum and Chi-Square tests respectively. Correlations of *Pf*-specific antibodies with VL and CRP were obtained using Spearman correlation. The study found that IgM, IgG1 and IgG3 levels against both AMA-1 and GLURP-R0 were significantly high in HIV infected individuals (*P*<0.0001). Antibody responses against AMA-1 were lower in individuals having CD4+ counts ≤200 cells/ml (*P*=0.01). Levels of IgM and IgG1 against both AMA-1 and GLURP-R0 were associated with CRP levels (*P*=0.01, 0.05, 0.02 and 0.004 respectively) and IgM and IgG1 against both antigens were associated with VL (*P*=<0.001, 0.02, 0.02 and 0.01 respectively). The data suggest that HIV infection leads to increased IgM, IgG1 and IgG3 responses, but low CD4+ counts are associated with lower total IgG and IgG1 responses. These findings provide an insight into a better understanding of malaria-specific antibody responses due to HIV infection. Future studies should assess the cellular mechanisms leading to increased antibody levels in HIV-malaria co-infection.