ABSTRACT

The prevalence of glucose-6-phosphate dehydrogenase (G6PD) deficiency and sickle cell trait (SCT) may vary within very close localities in malaria endemic areas due to dynamics in malaria incidence and demographics. Immunoglobulin-G (IgG) antibodies against *Plasmodium falciparum* (Pf) circumsporozoite protein (CSP) and apical membrane antigen-1 (AMA1) confer protection against malaria. Both G6PD deficiency and SCT increase resistance to Pf malaria and it is not well understood if this protection arise as a result of altered levels of IgG responses to these Pf antigens. The objectives of this study were to: determine the prevalence of SCT and G6PD deficiency; determine the association of SCT and G6PD deficiency with anti-CSP and anti-AMA1 IgG antibody responses in residents of Kanyawegi sub-location, Kisumu county of western Kenya. In this cross-sectional study, a total of 300 individuals from malaria endemic Kanyawegi sub-location participated. Venous blood was collected for laboratory assays, malaria parasite detection and enumeration. G6PD phenotyping was done using paper chromatography while PCR, restriction enzyme digestion and gel electrophoresis was used to genotype Haemoglobin-S (HbS). IgG levels against CSP and AMA1 were determined using indirect ELISA. Chi-square was used to compare the proportions of SCT and G6PD deficiency between groups. Comparison of IgG levels between groups was done using Mann Whitney U test. Association between anti-CSP and anti-AMA1 IgG antibody responses and different HbS and G6PD deficiency phenotypes was assessed using logistical regression analysis. Results showed that G6PD deficiency and SCT prevalence were 8.3% and 14.4% respectively and low frequencies of Anti-CSP IgG antibodies were significantly associated with G6PD deficiency [p=0.020] and SCT [p=0.016] but no influence on frequency of anti-AMA1 IgG antibodies by G6PD deficiency [p=0.819] and SCT [p=0.862]. However, in children with either SCT or G6PD deficiency, the malaria negative ones had significantly higher levels of anti-CSP IgG than the malaria positive ones [p=0.040; p=0.003]. This suggests that anti-CSP IgG antibodies collaborate with SCT and G6PD deficiency in protecting against paediatric clinical malaria but not in adults. Similar studies involving other genetic disorders like thalassemia need to be done to create a clear understanding of malaria immunogenetics. Information on prevalence of SCT and G6PD deficiency is important when planning for malaria chemotherapy and research in this area. Understanding humoral responses and these genetic disorders help elucidate knowledge on anti-malarial immunity and vaccine targets since the CSP-cased vaccine under trial elicit IgG antibodies which may give different findings in individuals with these genetic disorders.