ABSTRACT

An estimated 198 million malaria cases were reported worldwide of which 128 million were in African region while 16 million were reported in Kenya by the end of 2013. The prevalence of malaria in Vihiga, Kenya was 52% and this is greatly driven by *Plasmodium falciparum* drug resistance, ecological and agricultural factors supporting malaria transmission and vector proliferation. Influence of host genetic factors like blood group, glucose-6-phosphate dehydrogenase (G6PD) and haemoglobin genotypes on *falciparum* malaria outcome varies geographically and ethnically. However, their influences on *Plasmodium falciparum* infection among children in Vihiga, Kenya, had not been determined. Studies on these genetic variants and their influences on malaria outcome have been done in isolation but none had looked at their concurrent inheritence. This hospital based cross-sectional study, therefore, determined the influence of blood group, G6PD, haemoglobin genotypes and the concurrent inheritence of G6PD and Hb genotypes on malaria infection among children under 3 years in Vihiga, Kenya. A total of 414 malaria uninfected healthy controls and 440 malaria infected cases (severe malaria, n=72, and uncomplicated malaria, n=368) were recruited and their socio-demographic, clinical and laboratory information collected. About 2 microliters of whole blood was collected from each study participant and used for malaria microscopy diagnosis as well as haemoglobin estimation, G6PD genotyping, blood group. Determination of blood groups was done by forward grouping using commercial antisera, G6PD done by flourescent spot test Hb genotypes by alkaline electrophoretic scan and malaria tests were performed microscopically after staining the films by giemsa. The influence of blood group, haemoglobin and G6PD genotypes on malaria was determined using multivariate logistic regression analysis with malaria uninfected healthy control as reference group controlling for age and gender. Blood group A (P=0.600), B (P=0.227), AB (P=0.279) and O (P = 0.787) had no influence on uncomplicated malaria and severe malaria infection (P > 0.05). G6PD normal (P=0.770), intermediate (P=0.327) and deficient (P=0.309) showed no influence on uncomplicated malaria. However, G6PD normal children were 4.81 times more likely to suffer from severe malaria (odds ratio [OR], 4.81; [95% CI], 2.10-8.01; *P* =0.001) relative to G6PD intermediate (odds ratio [OR], 1.61; [95% CI], 1.09-2.73; *P* = 0.015) and deficient (odds ratio [OR], 0.23; [95% CI], 0.14-0.85; *P* = 0.034) children. Haemoglobin AA (P=0.551), AS (P=0.509) and SS (P=0.753) had no influence on uncomplicated malaria. There was no influence of haemoglobin AA (P=0.654), AS (P=0.721) and SS (P=0.831) on severe malaria. Concurrent inheritance of hemoglobin genotype with either G6PD normal, intermediate and deficient had no influence on uncomplicated and severe malaria (P>0.005). The findings of this study provide better understanding of genetic variants and their influence on malaria *P. falciparum* malaria outcome. Determination of G6PD deficiency provides an insight into the choice of ant malarial medicine and better clinical management of patients suffering from malaria.