Author: Baariu FK., Wigina RN

Institution: Technical University of Mombasa, Mombasa.

Background: Glucose-6-phosphate dehydrogenase deficiency (G6PDd) is an X-linked hereditary genetic effect that has been estimated to affect 400 million people worldwide. This deficiency is associated with hemolytic disorders depending on the molecular variants and exposure to hemolytic triggers such as anti-malarial drugs like Primaquine and foods such as fava beans. Symptoms can go asymptomatic unless triggered by oxidative stress. Transfusion of blood with this deficiency may result in complications including neonatal jaundice.

Methods: Methaemoglobin reduction test (MRT) was used to determine the occurrence of G6PDd in blood donors at the regional blood transfusion centre (RBTC)-Mombasa.

Results: Probability proportions and rank correlation were used to determine statistical significance of this enzyme deficiency. A total of 386 donor units were identified during the study. Out of these, 11.67% (35) were deficient with very low activity of G6PD and 13.2% (51) donors exhibited borderline enzyme activity. The ABO blood types showed a variance in the proportions with G6PDd. Blood type A had the highest number of donors with diminished G6PD activity. Of the 105 “A” donors, 11 were deficient representing 10.47%. Blood group B donors had the lowest probability with 5.88% having G6PDd. We did not find any correlation between low Hb and G6PDd.

Conclusions: G6PDd exists among healthy donors as either variants with slightly less than normal activity to those with very diminished activity. There is a higher probability that a blood group “A” person is more likely to have G6PDd in comparison to other ABO blood types. G6PD deficient individuals will not necessarily have a low haemoglobin level.