Hyperbilirubinemia in glucose-6-phosphate dehydrogenase-deficient male newborns in Al-Ahsa, Saudi Arabia

To the Editor

I read with interest the study by Al-Abdi et al[1] on the hyperbilirubinemia in glucose-6-phosphate dehydrogenase-deficient male newborns in Al-Ahsa, Saudi Arabia. The correlation between various hematological parameters and normal glucose-6-phosphate dehydrogenase (G6PD) level has been previously assessed. A statistically significant negative correlation was found between G6PD level and hemoglobin (Hb), packed cell volume (PCV), red blood cell (RBC) count, mean corpuscular hemoglobin (MCH), and mean corpuscular volume (MCV). A statistically significant positive correlation was found between G6PD level and white blood cell (WBC) count and reticulocyte count, but no significant correlation was found between G6PD level and mean corpuscular hemoglobin concentration (MCHC). The negative correlation between G6PD level and Hb suggests that anemic people have higher G6PD levels than normal individuals. The positive correlation between G6PD level and WBC count indicates that WBC may play an important role in contributing to the G6PD level.[2] Al-Abdi et al[1] addressed a unique observation in their study in which hyperbilirubinemia in G6PD-deficient Ahsai male newborns was characterized by higher levels of both hematocrit and Hb levels, and lower reticulocyte percentage compared to their non-hyperbilirubinemic counterparts. Concomitant Gilbert disease with G6PD deficiency was proposed by Al-Abdi et al[1] to partly explain that unique observation. Various studies have disclosed many factors that might ameliorate the hematological profile in persons with G6PD deficiency. For instance, ABO-incompatible/G6PD-deficient neonates, compared with those with either condition alone, are not at increased risk for hemolysis or hyperbilirubinemia.[3] Based on the prevailing various hemoglobinopathies and enzymopathies in Saudi Arabia, various studies demonstrated that co-inheritance of 2 or more abnormal RBC genes in the same individual might modify the severity of the clinical spectrum and/or hematological profiles.[4-6] In a recent study, both female heterozygotes and homozygotes of G6PD deficiency in association with different hemoglobinopathies showed reduced values of hematological indices: Hb, MCV, MCH, MCHC, and RBC count in comparison with normals. Red blood cell indices were found further reduced in male G6PD deficiency concurrence with hemoglobinopathies in homozygous condition, namely, sickle cell disease (HbSS), or hemoglobin E disease (HbEE). Hematological indices were significantly lower, except WBC counts and fetal Hb in male G6PD deficiency with co-existing homozygous sickle cell disease in comparison with counterpart sickle cell trait and normal controls.[7] Therefore, new studies in Saudi Arabia are warranted to ascertain the contribution of the aforementioned factors to the unique observation stated in Al-Abdi et al's study.[1]

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Reply from the Author

No reply from the Author.

References

1. Al-Abdi SY, Mousa TA, Al-Aamri MA, Ul-Rahman NG, Abou-Mehrem AI. Hyperbilirubinemia in glucose-6-phosphate dehydrogenase-deficient male newborns in Al-Ahsa, Saudi Arabia. ***Saudi Med J*** 2010; 31: 175-179.
2. Ajlaan SK, al-Naama LM, al-Naama MM. Correlation between normal glucose-6-phosphate dehydrogenase level and haematological parameters. ***East Mediterr Health J*** 2000; 6: 391-395.
3. Kaplan M, Vreman HJ, Hammerman C, Leiter C, Rudensky B, MacDonald MG, et al. Combination of ABO blood group incompatibility and glucose-6-phosphate dehydrogenase deficiency: effect on hemolysis and neonatal hyperbilirubinemia. ***Acta Paediatr*** 1998; 87: 455-457.
4. el-Hazmi MA, Warsy AS. The effects of glucose-6-phosphate dehydrogenase deficiency on the haematological parameters and clinical manifestations in patients with sickle cell anaemia. ***Trop Geogr Med*** 1989; 41: 52-56.
5. Awamy BH. Effect of G6PD deficiency on sickle cell disease in Saudi Arabia. Indian J Pediatr 1992; 59: 331-334.
6. el-Hazmi MAF, Warsy AS, Al-Swailem AR, Al-Faleh FZ, Al-Jabbar FA. Genetic compounds-HbS, thalassemia, and enzymopathies: spectrum of interactions. ***J Trop Pediatr*** 1994; 40: 149-156.
7. Balgir RS. Hematological profile of twenty-nine tribal compound cases of hemoglobinopathies and G-6-PD deficiency in rural Orissa. ***Indian J Med Sci*** 2008; 62: 362-371.