Neonatal Jaundice and Glucose-6-Phosphate Dehydrogenase Deficiency

S RASHEED A HAYEE Y LODHI *R AHMED*
Institute of Blood Transfusion Service, Lahore
Correspondence to Dr. Sohail Rasheed

One hundred new born males babies aged 7 days and above were included in this study. These were divided into two groups – Group I included G6PD normal subjects and Group II included G6PD deficient subjects. Total bilirubin and G6PD enzyme levels were done by commercially available kits. Results were analysed by using students 't' test and level of significance was done. A significant increase in total bilirubin level was observed in infants of G6PD deficiency, Erythrocyte G6PD level is significantly decreased in 06% of infants born with neonatal jaundice.
Key Words: Neonatal Jaundice, G6PD deficiency.

Glucose-6-phosphate dehydrogenase deficiency is a hereditary abnormality in which the activity or stability of the enzyme glucose-6-phosphate dehydrogenase is markedly diminished[1]. Glucose-6-phosphate dehydrogenase deficiency is the most common human enzymopathy [2]. Clinical and biochemical analyses have identified nearly 400 variants of G6PD[3]. There is a wide variety of normal genetic variants. The commonest being type B (Western) and type A in Africans [4]. The G6PD variant that occurs in erythrocytes of some people of the Mediterranean area is called G6PD Mediterranean. Its electrophoretic mobility is like that of G6PDB but its activity is markedly diminished to less than 05% to that of G6PDB. Several G6PD variants have been described in South East Asians, most common are the variant Mohidal and variant union. Although in many blacks the B gene is replaced by A gene, only those individuals with A⁻ genotype are subject to hemolytic episodes [5]. A large number of drugs and chemicals have the capacity to precipitate hemolytic reaction in G6PD deficient individuals e.g antimalarials, sulphonamides, sulphones, nalidixic acid, aspirin, antihelminthics, probenecid, Vit-K analogues The clinical features in G6PD deficient individuals are those of hemolytic anemia[6]. During hemolytic episode intravascular hemolysis rapidly develops with anemia. Hemolytic jaundice and hemoglobinemia. Between the crises the blood count is normal. The enzyme deficiency is detected by direct enzyme assay on the red cells. During crises the blood film may show contracted and fragmented cells, bite cells and blister cells. Heinz bodies may also be seen. Reticulocyte count also increases [4].

Methodology:
100 new born male babies aged 7 days and above were included. Six ml of blood was drawn by using 10cc syringe. Total bilirubin and erythrocyte G6PD levels were done by using commercially available kits. Results were analysed and level of significance was done[7].

Results:
Results and level of significance of these neonates are given in table 1.

Table 1: Total bilirubin and erythrocyte G6PD levels in normal and deficient subject

Tests	Group-I	Group-II	I vs II
Total bilirubin	9.4 ± 2.4	18.2 ± 5.3	p <0.01 (HS)
Erythrocyte G6PD level	133.3±16.7	82.2 ± 11.9	p <0.01 (HS)

HS (Highly significant)

Discussion:
Total Bilirubin Level:
In the present study, total bilirubin level was increased in infants of neonatal jaundice G_6PD deficiency (Group II) when comparing with subjects without G_6PD deficiency (Group I) and difference was highly significant ($p<0.01$) statistically. This increased bilirubin level in the infants of G_6PD deficiency may be due to hemolytic process going on in the body. These findings are consistent with the results of Beutler (1994)[8] who also observed hyperbilirubinemia in subjects of neonatal jaundice with G_6PD deficiency.
Erythrocyte G_6PD Level :
In the present study, G_6PD level was found to be reduced in 06 infants (6%) and the level was in normal limit in 94 infants (94%) and statistically difference was highly significant ($p<0.01$) statistically. These findings are in favour of the results of Ming et al (1992) [9], Kaplan and Abramov (1992) [10] and Beutler (1994)[8] who also observed 6 – 7 % deficient G_6PD infants in their study.

Conclusion:
Erythrocyte G6PD level is decreased in 06% of infants born with neonatal jaundice. Highly significant correlation was found between hyperbilirubinemia and erythrocyte G6PD deficiency in infants.

Reference:
1. William JW, Beutler E, Allan JE, Marshal A. Lichtmann Eds. Hematology 3rd ed. Singapore Mc Graw-Hill book Company 1991; 561-591.
2. Sodeinde O, Glucose-6-phosphate Dehydrogenase Deficiency. Baillieres-Clin-Hematol 1992; 5 (2): 367-82.
3. Chin D.T, Zuo L, Chao L. Molecular characterization of glucose-6-phosphate dehydrogenase deficiency in patients of Chinese descent and identification of new base

substitution in the human G6PD gene blood 1993; 81 (8): 2150-4.
4. Hoffbrand AV, Petit JE. Essential hematology 2nd ed. Singapore. PG Publishing 1993; 21-22 and 83-86.
5. Norbert W, Ph. D. Fundamentals of clinical chemistry. W.B. Saunders Company 2003; 666-669.
6. Dacie SJV, Lewis SM. Practical haematology, Investigation of Coagulation 9th Ed. Edinburgh: ELSB, Churchill Livingstone, 2001; 279-318.
7. Bland M. AN introduction in Medical Statistics Ist Ed. 1988; 112-133.
8. Beutler E: G6PD deficiency. Review; Blood 1994; 3613-36.
9. Ming WY, Kwang JH, Kuang DW, Chien JC. Association between G6PD deficiency and neonatal Jaundice. Int. J Epidemiology 1992; 21 (5): 947-952.
10. Kaplan and Abramov. Neonatal Hyperbilirubinemia associated with G6PD deficiency in Jewish neonates. Pediatrics 1992; 90(3): 401-405.