Introduction

Neonatal hyperbilirubinemia, (defined as a total serum bilirubin level exceeding 5 mg/dl) is a frequent problem as neonatal jaundice affects 60% of full-term infants and 80% of preterm infants in the first 3 days of life. Although it is a transient problem, the condition accounts for up to 75 % of hospital readmissions in the first week after birth ***(Porter and Dennis, 2002).***

The mechanism of neonatal hyperbilirubinemia is variable including: Bilirubin overproduction witch occurs in hemolytic diseases with either positive Coombs' test (ABO incompatibility, Rhesus incompatibility, and minor blood group antigens) or negative Coombs' test (red blood cell membrane defects, e.g.: spherocytosis, elliptocytosis, and/or red blood cell enzyme defects, such as glucose-6-phosphate dehydrogenase (G-6-PD) and pyruvate kinase deficiencies ***(Porter and Dennis, 2002).***

Sepsis and some drugs are other examples of hemolytic diseases. Bilirubin overproduction may occur in non- hemolytic diseases, like cephalhematoma, bruising, central nervous system hemorrhage, swallowed blood, polycythemia, ileal atresia, and pyloric stenosis. Breast milk associated jaundice also is an important factor contributing to indirect neonatal hyperbilirubinemia and it has two forms: early onset (breast feeding jaundice) and late onset (breast milk jaundice) ***(Siberry and Iannone, 2000)***.

Decreased bilirubin conjugation also occurs in physiological jaundice Crigler - Najjar Syndrome, hypothyroidism, sepsis and premature newborns ***(Dennery et al., 2009).***

G-6-PD is a crucial X-linked enzyme producing reduced glutathione in the erythrocyte cytoplasm for protecting hemoglobin

against oxidative damage. The presence of unopposed oxidizing agents leading to oxidation of the sulfhydryl bridges between parts of the hemoglobin molecule decrease the solubility of hemoglobin, leading to precipitations called Heinz bodies ***(Cappellini MD, Fiorelli G, 2008).***

The prevalence of G-6-PD deficiency among Caucasian populations ranges from less than one in 1000 among Northern European populations to 50 percent of the males among Kurdish Jews. G-6-PD deficiency is also found among certain Chinese populations and in Southeast Asia but it is rare in Japan. G-6-PD deficiency of the A-type is very common in West Africa, and the prevalence among African American males is approximately 11 percent ***(Beutler, 2006).***

Jecterus neonatorum in G-6-PD deficiency probably is due principally to inadequate processing of bilirubin by the immature liver of G-6-PD deficient infants, although shortening of red cell life span may play a role. Severe jaundice due to G-6-PD deficiency seems to be limited to infants who have also inherited a mutation of the Uridine - Di phospho - Glucuronic acid transferase -1 (UDPGT-1) gene promoter ***(Beulter, 2008).***

Some cases have been reported in G-6-PD deficient infants in Africa, but in the United States the incidence of jaundice in G-6-PD A-type dose not appear to be increased. The cause of relatively low incidence of neonatal jaundice in infants with G-6-PD A-type mutation is not clear. It could be due to the higher residual enzyme activity, but it dose not appear to be related to the incidence of the UDPGT-1 promoter mutation which is actually more common in Africans and less common in Asians than it is in Europeans ***(Beutler, 2006).***

Evaluation of G-6-PD deficiency should be considered, especially for infants who are older than four days, have appositive family history, or are of East Asian, Greek, Mediterranean, or African descent ***(Melton and Akinibi, 1999).***