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Glucose-6-Phosphate Dehydrogenase Deficiency in Neonatal Hyperbilirubinemia in a South Indian Referral Hospital

Kurien Anil Kuruvilla
Shaji T. Sukumar Atanu
Kumar Jana

Glucose-6-phosphate dehydrogenase (G-6-PD) is essential to maintain stability of red blood cells(1). The inherited deficiency of this enzyme may manifest as congenital

nonspherocytic hemolytic anemia, drug-induced hemolytic anemia or hemolytic disease of the newborn.

G-6-PD deficiency is the most prevalent enzyme deficiency worldwide. Routine screening of children and adults in various parts of India indicates that the prevalence

From the Neonatology Unit, Christian Medical College Hospital, Vellore 632 004, Tamil Nadu.

Reprint requests: A.K. Jana, Neonatology Unit, Christian Medical College Hospital, Vellore 632 004, Tamil Nadu.

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of G-6-PD deficiency ranges widely from as low as 0.2% to as high as 19%(2-8). But apart from the high prevalence noted in some ethnic groups(6,8), in most parts of the country the prevalence is less than 6%(2-7). Neonates with hyperbilirubinemia constitute a high-risk group in whom the prevalence of G-6-PD deficiency is likely to be high. However, there is no data from South India to indicate the extent to which G-6-PD deficiency contributes to neonatal hyperbilirubinemia. The present study was undertaken to ascertain the role of G-6-PD deficiency in neonatal hyperbilirubinemia in South India, to determine whether this subset of children need to be routinely screened for G-6-PD deficiency.

Subjects and Methods

This study was conducted among neonates with hyperbilirubinemia admitted in the Christian Medical College and Hospital, Vellore. From February to December 1996, we consecutively investigated all term babies with serum bilirubin levels more than 12 mg/dl ($>205 \mu\text{mol/l}$) and preterms with serum bilirubin exceeding 15 mg/dl ($<255 \mu\text{mol/l}$) for G-6-PD deficiency after ruling out ABO and Rh incompatibility by blood grouping, direct Coomb's test, reticulocyte count, and peripheral blood smear. They were also tested for hemoglobin concentration, total and direct bilirubin, random blood sugar, and G-6-PD levels. For the G-6-PD assay, 1 ml of anticoagulated blood in a glass bottle was sent and analyzed in the laboratory within two hours. Determination of the activity of G-6-PD was done by spectrophotometry(9). When low G-6-PD levels were encountered ($<120 \text{ U/L}$), the test was repeated on a fresh sample to confirm deficiency of the enzyme. Statistical analyses was done using the Chi-square and Z-test to compare differences between G-6-PD deficient and non-deficient babies.

Results

Two hundred and twelve neonates with hyperbilirubinemia not caused by ABO or Rh incompatibility were enrolled in the study. Twenty five (11.8%), were found to have deficiency of G-6-PD. All babies were born at Christian Medical College and Hospital, Vellore, except one who was born elsewhere and transferred with severe jaundice on the fourth postnatal day. Of the G-6-PD deficient neonates, 16 (64%) were males and 9 (36%) were females; 4 (16%) were preterm and low birth weight. The frequency of G-6-PD deficiency was similar in children of different birth weight, gestational age and religious background.

The hematological profile of neonates with hyperbilirubinemia who were investigated for G-6-PD deficiency is shown in *Table I*.

Jaundice was not detectable within the first 24 hours in any baby; the mean age of onset of jaundice was 61.4 (± 21.6) hours. Thirteen (52%) patients had a peak serum bilirubin level in the range of 15-20 mg/dl. Only the outborn baby transferred with severe jaundice had a serum bilirubin of 32 mg/dl and required an exchange transfusion; in all other cases jaundice improved with phototherapy alone. The mean duration of phototherapy was 3.4 (± 1.1) days. None of the babies developed features of kernicterus.

Discussion

Several studies done across India by screening of all newborns have shown that the prevalence of G-6-PD deficiency is less than 6%(2-7). But in ethnic groups such as the Parsees and Bhanushalis, it was as high as 16 and 19%, respectively(6,8). However, the studies done in Chandigarh, Delhi and Thailand investigating the contribution of G-6-PD deficiency to neonatal hyperbilirubinemia reveal a uniformly similar

TABLE I—Hematological Profile of Neonates with Hyperbilirubinemia

Parameter	G-6-PD deficient (n=25)	G-6-PD normal (n=187)
PCV (%)		
Mean (SD)	49.9 (7.5)	50.1 (4.8)
Retic Count (%)		
Mean (SD)	3.0 (1.5)	3.2 (1.8)
Nucleated RBC/100 WBC		
Mean (SD)	3.8 (2.2)	3.1 (1.0)
Blood smear suggestive of hemolysis (%)	36	21

None of the differences between the two groups were statistically significant ($p > 0.05$).

contribution of 12%(10-12). Similarly, in this study from South India, 11.8% of newborns investigated for hyperbilirubinemia were found to have G-6-PD deficiency.

A majority of G-6-PD deficient babies had features mimicking exaggerated physiological jaundice. The onset of clinical jaundice was always after 24 hours, the peak serum bilirubin rarely exceeded 25 mg/dl, and there was almost universal response to phototherapy alone. Only by investigating the G-6-PD level was it possible to make a diagnosis of G-6-PD deficiency, in a population where this disorder has not been well recognized earlier.

The incidence of G-6-PD deficiency among infants with hyperbilirubinemia is a measure of the public health aspect of the problem. Thus, in developing countries like India, where the prevalence of G-6-PD deficiency varies widely in the large population, routine screening of all newborns is not feasible or cost-effective except in selected ethnic groups where a high prevalence of G-6-PD deficiency has been reported. Yet the importance of identifying G-6-PD deficiency in the newborn period cannot be overlooked so that appropriate counselling is provided to the family and

potentially harmful drugs are not administered to these infants and their nursing mothers. Thus, we would recommend that a more appropriate strategy would be to identify those with G-6-PD deficiency by focusing our efforts on neonates with significant jaundice.

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Clinico-Hematological Profile of Megaloblastic Anemia

Sunil Gomber
Kusum Kela
Neelam Dhingra

Megaloblastic Anemia is one of the important causes of anemias in children. It is not an infrequent entity in poor socio-economic condition. This condition has protean manifestations in childhood, sometimes mimicking a hematological malignancy like leukemia. Diagnosing this dis-

ease assumes great clinical importance since it responds exceedingly well to treatment. The present study evaluates the varying clinico-hematological manifestations in 29 patients diagnosed as megaloblastic anemia over a three year period.

Subject and Methods

Twenty nine children (age range 3Vi months to 12 years) diagnosed as megaloblastic anemia over a period of three year (March 1993 to March 1996) were prospectively studied. All anemic children admitted with or without bleeding manifestations had their peripheral blood smear examined. Complete hemogram including platelet count and mean corpuscular volume (MCV) were also carried out in each child using Coulter T860 particle counter. The platelet count obtained from Coulter counter was always confirmed by peripheral smear examination. Cases with macrocytic blood picture on smear examination were subjected to bone marrow examination to confirm the diagnosis of

From the Departments of Pediatrics and Pathology, University College of Medical Sciences and Guru Tegh Bahadur Hospital, Shahdara, Delhi 110 095.

Reprint requests: Dr. Sunil Gomber, F-4(A) Vijay Nagar, Delhi 110 009.

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