Hyperbilirubinaemia and erythrocytic glucose 6 phosphate dehydrogenase deficiency in Malaysian children

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Summary

Cord blood from 8,975 babies delivered in Hospital Sultanah Aminah Johor Bahru over a period of eight months (1st August 1985 to 31st March 1986) were screened for G6PD deficiency. The overall incidence was 4.5% in Chinese, 3.5% in Malays and 1.5% in Indian babies.

One hundred of these babies were observed in the nursery for seven days and their daily serum bilirubin recorded. The serum bilirubin peaked at 96 hours to a value of 12mg%. None of the babies in the nursery developed a serum bilirubin level of more than 15mg%. Six of the babies with G6PD deficiency that were sent home were readmitted with hyperbilirubinaemia that needed exchange transfusion.

Key words: Cord blood, G6PD deficiency, hyperbilirubinaemia, exchange transfusion.

Introduction

Human red cell glucose-6-phosphate dehydrogenase (G6PD) represents less than one part in 20,000 of the protein of the red cell.[1] Yet deficiency of this enzyme can cause severe neonatal jaundice that can lead to kernicterus and deafness. In 1964, 43.7% of kernicterus in Singapore were caused by G6PD deficiency.[2] In Malaysia, G6PD deficiency is still one of the leading causes of kernicterus in the newborn period. The purpose of this paper is to study the incidence and morbidity of G6PD deficiency in the three major races – Malays, Chinese and Indians in Malaysia.

Materials and method

A total of 8,975 newborns in Sultanah Aminah General Hospital, Johor Bahru that were delivered between 1st August 1985 and 31st March 1986 were studied. Cord blood taken at birth were smeared on a triangular piece of filter paper to a size of a 20 cents coin and delivered to the laboratory within six hours. In the laboratory, these blood samples were screened for G6PD using the modified Beutler flourescent spot test.[3,4]

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The results of the screening were reported by phone to one of the authors who interviewed the mothers in the post natal ward before they were discharged. The mothers were given a brief explanation on G6PD deficiency, neonatal jaundice and advised to avoid haemolytic triggers like mothballs and Chinese herbs. She was also advised to bring the child immediately to the nursery should the child develop jaundice. In addition she was also given a handout on G6PD deficiency.

One hundred G6PD deficient babies were chosen at random and admitted to the nursery for observation. These were healthy term babies with no evidence of isoimmunisation and cephalhaematoma. In the nursery, these babies were nursed in open cots. The illumination in the nursery averaged 4.0uW/cm^2 in the 400 to 500m range. Measurements were made by the radiometer PRIII/ Narco Scientific (Air Shields Division). None of these babies were exposed to direct sunlight. Serum bilirubin levels were monitored daily in the morning for seven days. Capillary blood samples for bilirubin estimation were kept in a dark envelope until estimation. Bilirubin levels were determined by Bilmeter-D, Mochida Luketon Model MEB – 332. These babies were discharged after seven days.

The babies who were not admitted for observation were reviewed by one of the authors when they developed jaundice. Those with serum bilirubin levels of more than 15mg% were admitted for further investigation and treatment.

Results

In the eight months period, a total of 8,975 babies were screened for G6PD deficiency. The results are summarised in Table I. The overall incidence was highest amongst the Chinese babies where 121 (4.5%) out of 2,643 babies screened were found to be G6PD deficient. Next was the Malay babies in whom 196 (3.5%) were G6PD deficient out of the 5,557 screened. In 775 Indian babies screened, 11 (1.5%) were found to be deficient.

Figure 1 shows the serum bilirubin levels of the 100 G6PD babies that were observed for seven days. On day one, the serum bilirubin in all the three racial groups were high – 6mg% (3.8–

Table I

Incidence of G6PD deficiency in infants delivered in Hospital Sultanah Aminah Johor Bahru (August 1985 – March 1986)

Ethnic Group	No. Tested	G6PD Deficiency (%)				
		Intermediate		Deficient		Overall
		Male	Female	Male	Female	
Malay	5557	6 (0.1)	10 (0.2)	134 (2.4)	46 (0.8)	196 (3.5)
Chinese	2643	2 (0.1)	9 (0.3)	79 (2.9)	31 (1.2)	121 (4.5)
Indian	775	0 (0.0)	2 (0.3)	6 (0.8)	3 (0.4)	11 (1.5)
Total	8975	8	21	219	80	328

11.6mg%). This level rises rapidly to a peak of 11mg% (4.0–15.2mg%) on the 4th day. The serum bilirubin levels of the three races are similar. The high peak of the Indian group on the 4th day may not be representative as the sample is too small.

Two hundred and twenty-two G6PD deficient babies were discharged home with adequate verbal and written advice. In this group, six were readmitted because of jaundice. On admission, an exchange transfusion was done for each of the six patients because their bilirubin levels were more than 20mg%. None of the infants developed kernicterus. In three of these infants, neonatal sepsis was the precipitating cause of the haemolysis while no precipitating cause could be found in the remaining three. None of the babies in the nursery had serum bilirubin levels of more than 20mg%.

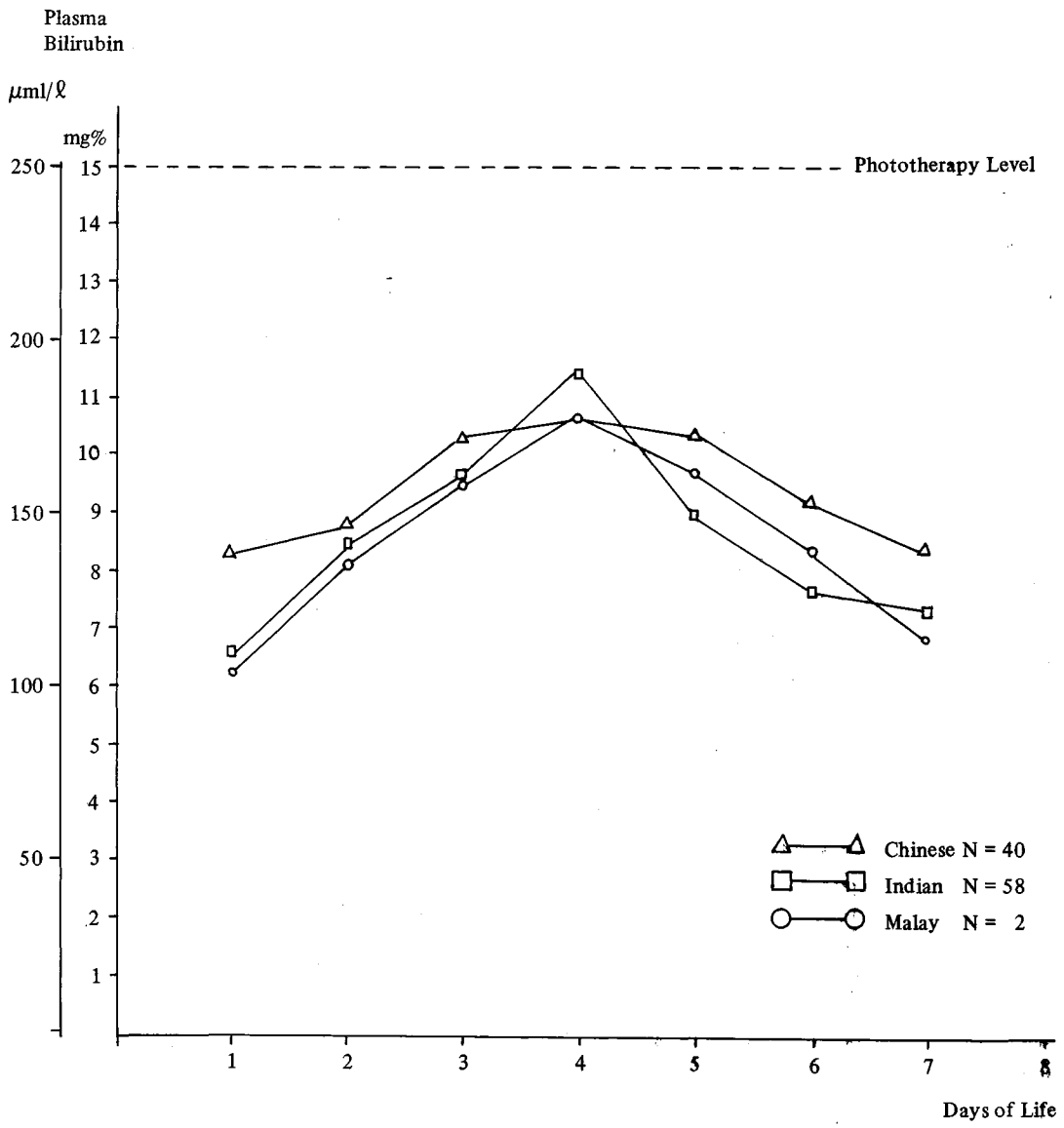

Fig. 1. Bilirubin levels of G6PD deficient infants of Malay, Chinese and Indian ethnic origins.

Discussion

The incidence of G6PD deficiency in the Chinese, Malays and Indians in Malaysia and Singapore has been the subject of many studies.[5,6,7] In 1975, a study done on 2,047 infants in Kuala Lumpur and Petaling Jaya showed the incidence amongst the Chinese to be 3.1%, the Malays 1.4% and 0.2% in the Indians. Another study in Malacca in 1984 on 15,300 infants showed the incidence in Chinese to be 3.8%, Malays 3.3% and in the Indians 1.5%.[5] The screening method used in these two studies were the modified Beutler fluorescent spot tests. Our study in Johore Bahru shows an incidence of 4.5% in Chinese, 3.5% in Malays and 1.5% in the Indians (Table II). The high incidence in the Chinese and the Malays make these two races at risk for kernicterus and any preventive program should be aimed at these two at risk groups.

Table II

Incidence of G6PD deficiency

Study	Present Study	Harcharan Singh		Robinson, Lau Lin & Chan	Wong
Year	1985/86	1983	1984	1975	1975
Total No.	8,975	12,579	15,300	2,047	286,802
Malay	3.5%	2.3%	3.3%	1.4%	2.2%
Chinese	4.5%	3.2%	3.8%	3.1%	1.7%
Indian	1.5%	1.3%	1.5%	0.2%	0.2%

The serum bilirubin level of G6PD deficient babies in the nursery were high on the first day of life, showing evidence of haemolysis. This compares well with the study of Tan.[8] The serum bilirubin peaks to a level of 10–12mg% on the 4th day of life in all three races, and then it begins to decline.

This study shows that there is some degree of haemolysis in G6PD deficient babies even in the absence of known haemolytic agents. Spontaneous haemolysis in deficient infants who have not been exposed to any known haemolytic stress has been reported elsewhere.[9] There is no significant differences in the degree of haemolysis amongst the three races studied.

Six of the G6PD deficiency babies discharged on the 1st day of life developed severe jaundice at home and were readmitted. All those babies needed exchange transfusion. The predisposing factors in three of them were found to be neonatal sepsis and no cause was found in the other three.

In our study none of the babies in the nursery developed serum bilirubin levels of more than 20 mg%. In the nursery, the linen and the clothing worn by the babies were supplied by the hospital. There were no risk of the clothings being exposed to mothballs. There was also no danger of the child being bathed in Chinese herbal preparations (San Chi, Chuan Lian) or given herbal tonics. Infections can be detected and treated early and phototherapy can be given when indicated. Hence in a controlled environment, G6PD deficient babies do not develop severe hyperbilirubinaemia. A similar study done in Singapore also confirmed these findings. In that study, it was found that either hyperbilirubinaemia occurs in the first week or not at all.[8]

In our study we found that all babies who developed severe hyperbilirubinaemia showed clinical evidence of jaundice before the third day of life and needed exchange transfusion before the 5th day of life. (Table III) None of the other discharged babies in the study were admitted after five days of life.

Table III

Incidence of G6PD deficiency

Study	Present study	Harcharan Singh		Robinson, Lau Lin & Chan	Wong
Year	1985/86	1983	1984	1975	1975
Total No.	8,975	12,579	15,300	2,047	286,802
Malay	3.5%	2.3%	3.3%	1.4%	2.2%
Chinese	4.5%	3.2%	3.8%	3.1%	1.7%
Indian	1.5%	1.3%	1.5%	0.2%	0.2%

Conclusion

G6PD deficiency is common in multiracial Malaysia. It has the highest incidence in the Chinese followed by the Malays and Indians. There is a mild haemolysis in all G6PD deficient babies at birth. The serum bilirubin peaks on the 4th day and then declines. In a controlled environment these G6PD deficient babies do not develop severe Hyperbilirubinaemia. We recommend that all G6PD deficient babies be observed in the nursery for at least five days after delivery.

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