

Health status of glucose-6-phosphate dehydrogenase (G6PD) deficient and other newborns in Sarawak, Malaysia

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ABSTRACT

Introduction: To study the health status of 200 G6PD enzyme-deficient Sarawak newborns, we compared their birth records to those of non-deficients in terms of low birth weight (LBW). To characterize ethnic groups, we analysed newborns in terms of LBW and G6PD population frequency.

Methods: For this retrospective study, health records for the year 2006 were obtained from the Sarawak General Hospital. Enzyme deficiency was determined by the fluorescent spot test.

Results: In a sample of 1248 newborns, 12.3% had low birth weight (LBW). The enzyme-deficient newborns were comparable to non-deficients in terms of LBW. Ethnic groups differed in their population frequency of the genetic deficiency. Genetic-deficiency data on Chinese and Melanau in Sarawak are reported here for the first time. In contrast to the usual studies on adults, G6PD studies on newborns reveal the deficiency transmission rate and provide a baseline to gauge the future health status of deficient children.

Conclusion: While deficient newborns showed no relative excess of LBW, the deficiency persists in populations in Sarawak, despite the known vulnerability to hemolytic anaemia caused by oxidizing agents. This persistence accords with the persistence of malaria in Sarawak, to which G6PD deficiency is known to provide some resistance.

Keywords: Sarawak, newborns, G6PD, health, genetics

INTRODUCTION

We studied the newborn records in Sarawak General Hospital in Kuching for the year 2006 and report the distribution of G6PD deficiency in Sarawak ethnic groups as well as the relationship between low-birth-weight (LBW) and G6PD status. It is known that LBW is an important detriment to infant growth and survival worldwide and that G6PD deficiency is implicated in resistance to malaria. In contrast to the usual studies on adults, studies on G6PD deficient newborns, while rare, provides a more accurate estimate of the deficiency transmission rate and provide a baseline for assessing the health status of deficient children.

METHODS

This retrospective study of newborns for the year 2006 was carried out at the Sarawak General Hospital using Labour Room and Record Unit data. Data were analyzed statistically using SPSS software.

RESULTS

Because the hospital records were incomplete on the incidence of prematurity, we analyzed LBW as an indicator of health status. To determine LBW and the newborn sex ratio, we sampled 1283 consecutive births, dating from 01-01-2006 to 14-02-2006. There

were 604 singleton and 3 sets of twin female births (610 total) and 655 singleton and 9 sets of twin male births (673 total). The male/female ratio was 1.10 (52.46% males). The M/F ratio by ethnicity had a range of 50.9% to 53.3%.

LBW was defined as a birth weight of ≤ 2.5 kg. Since a few records lacked birth-weight information, our data base totaled 654 males and 594 females, including 21 newborns with G6PD deficiency. Among males, 10.9% had LBW and among females, 13.8% had LBW, or 12.3% in total. For singleton births only, male LBW was 9.4% and female LBW was 12.8%. LBW in ethnic groups ranged from 7.9% in Chinese to 16.3% in Iban. In this sample of 1248, 2.7% had a birth weight of ≤ 2.0 kg., with Bidayuh having the highest frequency (4%).

G6PD deficiency was determined at birth by the fluorescent spot test. G6PD-deficient newborns totaled 200 out of 11220 live births at the hospital in 2006. Of these 200, 2 males had no reported birth weight. Among 191 G6PD-deficient males having birth-weight information, 21 had LBW (11.0%). Among G6PD-deficient females, 1 of 7 had LBW (14.3%). The findings on birth weight, taken all together, suggest that the deficient newborns were not relatively underweight. However, we also did a case-control analysis on LBW, pairing each deficient newborn with a non-deficient of the same sex and ethnicity born approximately the same time. Deficient twins were paired with non-deficient twins. The chi-square test showed that deficient newborns were not different from their peers in LBW frequency ($p=0.878$ with continuity correction).

In the census year 2000, the state of Sarawak had 22% Malays, 29% Iban, 26% Chinese, 8.0% Bidayuh, 5.5% Melanau and 10% others, but the population of the area served by the General Hospital, while unknown, is considered to differ from that of the state as a whole.⁽¹⁾ One possible indication of this is that among the 11220 hospital births in 2006 46.5% were Malay, 17.8% Iban, 17.4% Chinese, 13.4% Bidayuh, 1% Melanau, and about 4% other groups. Males in our 1283 sequential sample gave similar results: 47.9% Malays, 17.0% Iban, 16.7% Chinese, 14.0% Bidayuh, 1% Melanau, and 3% others.

Frequencies for G6PD deficiency are based on the percentages in males because the gene is on the X chromosome. Given 52.46% males in our sample of 1283 births and given 11220 births in 2006, approximately 5885 were male. Since both twins in two sets were deficient and thus possibly identical, we used 191 as the total for deficient males and 5883 as the total of male genomes. The proportion of deficient males was then 3.42%, but further analysis revealed some differences among ethnic groups. Based on the M/F ratio by ethnicity, the G6PD-deficient frequency was 3.5% for Malays, 3.4% for both Iban and Chinese, 2.2% for Bidayuh, and approximately 7% for Melanau, based on our sample of 4 out of 55 male Melanau being deficient. This is the first report on Chinese and Melanau frequencies for Sarawak.

DISCUSSION

We found 12.3% LBW. The state-wide average may be lower, however, as women in higher-income families can afford private medical-centre delivery. While G6PD deficient newborns in our study were like non-deficients in health status as measured by LBW, heterozygous mothers have some difficulties in pregnancy, leading to a relatively high proportion of unsuccessful events in the first trimester, a factor that may impinge especially on the survival of their enzyme-deficient male issue.⁽²⁾

Two other reports exist on LBW in Sarawak. One stated that the Sarawak average had dropped below 12% LBW by 1988, with Chinese having the lowest rate (6.7%) and Iban having the highest, at 13.6%.⁽³⁾ In contrast, our finding of 16.3% LBW in Iban newborns suggests continued health problems in this group. The second report stated that in the rural Bidayuh village of Mujat in the 1990's, 18% of the preschool children of the young parents studied had had LBW, while even more of the children had protein-calorie malnutrition.⁽⁴⁾ However, our more urbanized sample gave 13% LBW for Bidayuh newborns.

Our finding of low frequencies of G6PD deficiency in Sarawak ethnic groups differs somewhat from those of reports published a generation ago, which found male frequencies of 4% in Iban, 5% in Bidayuh, and 12% in Malays in Sarawak.^(5,6) Based on genetic equilibrium theory, we would have expected to find over 300 female newborns heterozygous for G6PD deficiency, but we found only 7 deficient females. Therefore, we did not estimate female frequencies of G6PD deficiency. While they are known to be underreported in Malaysia,⁽⁷⁾ a new screening method that detects heterozygotes has recently been developed that may rectify this problem.⁽⁸⁾

In terms of LBW, G6PD-deficient newborns in our study had the same health status as non-deficients, but older deficient children are uniquely vulnerable to hemolytic anemia upon ingestion of a variety of oxidizing agents. In addition, G6PD deficiency has been shown to protect against falciparum malaria,⁽⁹⁾ as do a few other genetic variants.⁽¹⁰⁾ Malaria has been on the rise in Sarawak, exceeding 3000 cases in 2001, with Malays, Chinese, and Melanau having the lowest incidence, Iban and Bidayuh moderate incidence, and Penan having by far the highest; nevertheless, the case load did decrease after 2001.⁽¹⁾ Long-term malaria pressure thus may account for the persistence of G6PD deficiency at polymorphic frequencies in Sarawak.

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