Onset of Bilirubin Rise in G6PD Deficient Neonates–Prenatal or Postnatal?

In utero rise of total serum bilirubin (TSB) occurs if the rate of fetal TSB production exceeds the rate of elimination by the maternal circulation, as in Rh iso-immunization. Recent reports indicate that impaired hepatic conjugation is a more important cause of neonatal jaundice in G6PD deficiency, than hemolysis due to post-natal oxidative stress(1,2). This raises the possibility of TSB rise starting in utero in G6PD deficiency. Kaplan *et al*(3) reported that the TSB levels in G6PD deficient neonates are higher within 3 hours of life compared to their healthy counterparts [2.9 ± 0.7 mg/dL vs 2.6 ± 0.6 mg/dL (P <0.05)](3). They concluded that TSB rise among G6PD deficient patients most likely starts in utero, but in the absence of cord bilirubin data, this conclusion could not be accepted with certainty.

To address this issue, we performed a case-matched cohort study. Cord blood samples of babies >34 wks and >1800 g were screened for G6PD deficiency(4), TSB and hematocrit (Hct). Congenitally malformed babies, Rh isoimmunized babies, those exposed to antenatal phenobarbital and whose parents refused consent were excluded. To identify a difference in mean cord TSB of 0.3 mg/dL (SD 0.5 mg/dL with an a error of 5% and 90% power, we recruited 44 cases and 88 controls, who were matched for gestation and birth weight (± 50 g). The cases were G6PD-deficient patients, and controls were randomly selected from among the matched subjects available in the non-deficient group. The prevalence of maternal diabetes, distribution of maternal ABO blood groups, use of oxytocin in labour, mode of delivery and Apgar score at 1 minute were similar in the cases and controls (all P >0.05). The mean cord TSB of cases did not differ significantly from controls [1.26 ± 0.8 mg/dL vs 1.18 ± 0.6 mg/dL respectively, P = 0.52]. Cord Hct of cases was also similar to controls [48.8 ± 9 vs 48.9 ± 6 respectively, P = 0.94]. Our study negates the possibility raised by Kaplan, *et al.* that in G6PD deficient neonates jaundice commences most likely in utero.

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