TRANSFUSION-RELATED DECREASE IN FETAL HEMOGLOBIN - A STRONG PREDICTOR OF SEVERE MORBIDITY IN VERY PRETERM INFANTS

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Fig 1

Fig 2

Background and Aims

Fetal hemoglobin (HbF) is a central determinant of oxygen exposure in fetal development. Transfusions with adult donor blood have been associated with morbidity in preterm infants. We hypothesized that the resulting decrease in fetal hemoglobin (HbF) is related to severe morbidity in very preterm infants.

Method

452 inborn infants < 30 GW admitted to the NICU in Lund 2009-15 with a mean (SD) GA of 26.0 (1.8) weeks and BW 876 (272) g. Data including HbF (%) were retrieved from 28 642 arterial blood gases analyzed during postnatal days (PND) 1-14. Development of broncho-pulmonary dysplasia (BPD), severity of retinopathy of prematurity (ROP) and necrotizing enterocolitis (NEC) were prospectively registered.

Results

Mean (SD) HbF (%) decreased from 84 (11) at PND 1 to 27 (15) at PND 14 in the total population (Fig 1). Decrease in HbF was correlated to administered donor blood (ml/kg), $R=0.96$, $p<0.001$. Infants who developed BPD (46 %), any ROP (23 %) and NEC (5 %) had lower mean (SD) HbF during the first postnatal week 61 (19), 58 (17) and 55 (16), than surviving infants without these morbidities, 79 (14), 74 (18) and 69 (20), all $p<0.001$. Differences remained significant after adjustment for GA, gender, total Hb, aPO₂ and aSatO₂. Mean (SD) HbF in relation to postnatal age and development of BPD is given in Fig. 2. Mean HbF during postnatal week 1 grouped in quartiles in relation to development of bPD is illustrated in Fig. 3.

Fig 3

Conclusion

A decreased proportion of HbF during PND 1-14 is highly correlated to severe morbidity in very preterm infants. Causal mechanisms may involve augmented tissue oxygen exposure or adverse effects of adult Hb