PARENTERAL SELENIUM SUPPLEMENTATION IN PRETERM INFANTS - A PILOT STUDY

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The essential trace metal selenium (Se) is a component of the enzyme glutathione peroxidase (GSH Px). This enzyme is part of the antioxidant defense system which protects against cellular damage due to oxygen toxicity by inactivating free radicals. Hyperoxia associated with oxygen therapy increases free radical production. Preterm infants are at risk of Se deficiency because of poor stores and low intake. Infant formulae have less than half the Se content of breast milk, and unsupplemented parenteral nutrition (PN) has none. There is evidence that prolonged PN results in Se depletion and this may be associated with increased incidence of chronic lung disease in preterm infants, particularly those who require significant oxygen therapy (Lockitch et al. 1989).

The aim of this preliminary study was to provide a profile of plasma Se levels in preterm infants in Adelaide as a pilot for a study to evaluate the need for, effectiveness and safety of Se supplementation in preterm infants receiving parenteral nutrition.

Plasma Se was measured in 42 infants who were divided into groups according to their primary source of nutrition - breast milk (BF; n=16), formulae (FF; n=9), unsupplemented PN (PN-SE; n=12), PN supplemented at 2ug/kg/day (PN + SE; n = 5) . Infants had a range of postnatal ages (PNA; median 23 days, range 3-96) and gestational ages (GA; median 30 weeks, range 26-41). Plasma Se was also measured in 44 adults and from cord bloods of 10 full term infants. Adult levels (82±17 μg/l) were significantly higher than cord levels(58±10 μg/l) which were also significantly higher than infant levels (27±10 μg/l, $P < 0.05$) . However there was no difference between the infants groups, including the PN+SE and the PN-Se groups. Plasma Se levels in the preterm infants were low with five infants having levels ≤ 13ug/1, similar to those seen in Se deficiency; (Keshans Disease). Further plasma Se declined significantly with PNA ($r=-0.466$ $P<0.01$). When the mean Se levels were adjusted for PNA as a covariate, levels in the BF group were significantly higher than the PN-SE group (32 vs 22 μg/l, $P < 0.05$) . Erythrocyte GSH Px was measured in 22 infants. There was no difference in GSH Px levels between the groups. As reported elsewhere the correlation of erythrocyte GSHPx with plasma Se found in Se depleted adults was not seen in any infant goup.

These results suggest (a) plasma Se levels are low in preterm infants, (b) plasma Se levels decline with PNA and therfore studies of Se status in infants need to control for PNA and GA and (c) supplementation with 2ug/kg/day may not be adequate to prevent biochemical depletion.

LOCKITCH, G., JACOBSON, B., QUIGLEY, G., DISON, P. and PONDRAY, M. (1989). J.Pediatr. 114: 865.

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